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UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF CALIFORNIA
SAN FRANCISCO DIVISION

IN RE: BABY FOOD PRODUCTS LIABILITY LITIGATION	Case No. 24-MD-301-JSC MDL No. 3101 Hon. Jacqueline Scott Corley
This document relates to: ALL ACTIONS	**PLAINTIFFS' OMNIBUS OPPOSITION TO DEFENDANTS' MOTIONS TO EXCLUDE**

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INTRODUCTION

There is overwhelming evidence that Defendants sold baby food products containing excessive levels of lead and arsenic. For example, in 2017 Plum sold a batch of Just Sweet Potato pouches marketed for infants "4 months & up" that contained [redacted]. Ex. 94, Plum Testing Results (MDL3101_PLUM_00002173). This is 7 times greater than the FDA current limit (10 ppb). Every baby that ate this pouch consumed 6.04 µg of lead in each serving. That dose, from a single pouch, is two and half times the U.S. Food and Drug Administration's ("FDA's") current *daily* Interim Reference Limit ("IRL") of 2.2 µg—a "benchmark in evaluating the potential for adverse effects of dietary lead exposure, such as the potential for neurodevelopmental effects." Ex. 15, FDA, *Action Levels for Lead in Processed Food Intended for Babies and Young Children: Guidance for Industry* (2025). In another example, Nurture sold a batch of "Blueberry Purple Carrot" yogis that tested for 641 ppb of lead. Ex. 95, Nurture Test Result (NURTURE-BF-MDL_00184296); Ex. 96, Laraway Dep. (Feb. 23, 2023) at 803:22-804:3 ("**Q.** [redacted] **A.** [redacted] **Q.** [redacted] **A.** [redacted]"). Every baby that ate this yogi snack consumed more than double the FDA's IRL *in each serving*. If they consumed the whole bag (which roughly fills an average toddler cup), it would be almost 18 µg of lead—which does not include lead exposure from other baby foods or the environment. Defendants knew their products contained these excessive levels of lead because they tested them. That testing, however, did not stop them from selling the products, knowing that babies would consume them.[1]

Pitted against the Defendants' conduct is a scientific consensus—early life ingestion of lead and arsenic harms neurodevelopment. Infants are uniquely susceptible to the neurotoxic effects of lead and arsenic in baby food. They consume more food by body weight, absorb more metals in their undeveloped gastrointestinal tract, have a permeable blood-brain barrier, and are undergoing rapid neurodevelopment. FDA was clear when it issued its final guidance about lead *in baby foods*: "Even low lead exposure can harm children's health and development, specifically the brain and nervous system." Ex. 15, FDA Lead Guidance (2025) at 4. It also was clear about arsenic *in baby foods*:

[1] Thankfully, Defendants started cleaning up their act. For example, Plum no longer sells a Just Sweet Potato pouch product. Ex. 102, Plum Website Screenshot. Similarly, Nurture's Blueberry Purple Carrot yogis are down to 2.95 ppb of lead. Ex. 103, Nurture Website Screenshot.

"FDA's risk assessment shows that inorganic arsenic exposure during … infancy[] and childhood may contribute to neurodevelopmental effects." Ex. 4, FDA, *FDA Supporting Doc for Action Level for Inorganic Arsenic Rice Cereal for Infants* (Aug. 1, 2020) at 6. Indeed, Defendants publicly acknowledge these neurodevelopmental effects outside of Court.[2]

Of these neurodevelopmental effects, two stand out. Hundreds of peer-reviewed studies confirm that exposure to lead and arsenic in early life increases the risk of developing autism spectrum disorder ("ASD") and attention-deficit hyperactivity disorder ("ADHD"). The scientific evidence is so robust that independent scientists openly state that lead and arsenic can cause ASD and/or ADHD in peer-reviewed literature. Although these peer-reviewed studies do not specifically limit their analysis to exposure from food, they necessarily capture those effects by focusing on a child's biomarkers of blood, urine, hair, teeth, and brain tissue. Infants and children exposed to greater amounts of lead and arsenic in early life are more likely to develop ASD and ADHD.

The general causation question, here, is whether ingesting the lead and arsenic to which an infant could be exposed from Defendants' baby foods can cause symptoms that would lead to a diagnosis of ASD or ADHD and/or exacerbate those conditions. To answer that question, Plaintiffs present epidemiologists (Drs. Ritz, Hu, and Gardener), toxicologists (Drs. Guilarte and Aschner), a neurologist (Dr. Shapiro), an exposure scientist (Dr. Jones), a pediatric nutritionist (Ms. Barr), and a geneticist (Dr. Reed). The qualifications of these experts are unassailable—indeed, many of them have spent their careers studying heavy metals' harmful effects on neurodevelopment, with multiple experts having published about the topic prior to being retained as experts.

The question, for this motion, is whether the methodology used and opinions reached by Plaintiffs' experts, more likely than not, meet the elements of Rule 702. To be clear, the question is *not* whether these experts are correct—that is the ultimate question for the fact finder. Rather, the question is whether these experts considered reliable materials, utilized reliable methodologies, and reliably applied those methodologies in reaching their opinions. And as evidenced by Plaintiffs'

[2] Defendant Sprout recently recalled multiple products due to heavy metal contamination, announcing publicly that "[e]exposure to lead, even at low levels, may increase BLLs," and that "[i]f a child is exposed to enough lead for a protracted period of time, this can affect learning and development or cause other long-term health problems." Ex. 97, FDA, *Sprout Organics Expands Voluntary Recall of Sweet Potato Apple and Spinach to Include Additional Lot Codes* (Sept. 23, 2025).

experts' detailed expert reports—citing and reviewing hundreds of studies—and their unmatched professional expertise, the weight of the evidence clearly tips toward admissibly.

Defendants make "kitchen sink" challenges to Plaintiffs' experts. They are each addressed. But, generally, these challenges do not attack the elements of Rule 702. Rather, they attempt to reframe the general causation question, misstate what Plaintiffs' experts actually did or said, and challenge the science directly. Defendants invite this Court to don a lab coat and wade into scientific debates. And, while the Court will surely have to engage on core scientific issues to properly adjudicate Defendants' Rule 702 motions, what the Defendants have done here is a step too far. For example, Defendants ask this Court to decide, as a matter of law, that whole swaths of peer-reviewed scientific evidence are unreliable and should not be considered. *See infra* Part III.D-F. To accommodate this request, the Court would need to decide what is good peer-reviewed science (and what is not) and then dictate what epidemiologists and toxicologists should be considering in assessing causation. That is not gatekeeping; that is scientific adjudication.

As presented in this omnibus opposition, Plaintiffs' experts each satisfy the elements of Rule 702. The Court should deny Defendants' Motions to Exclude in their entirety and order the Parties to prepare a bellwether trial process. It is time for this case to proceed with full discovery and trial.

BACKGROUND

As seen more fully in Plaintiffs' expert reports and the underlying evidence, the causal connection between heavy metal exposure – including through baby food – and ASD/ADHD is clear. So clear, in fact, that it has already led to government action at the federal and state levels.

I. Legislative and Regulatory Action Regarding Lead and Arsenic in Baby Food

The primary responsibility for assuring the safety of baby foods in the United States rests with the baby food manufacturers. *See* Ex. 1, FDA, *Ltr. to Baby and Toddler Food Manufacturers and Processors* (March 5, 2021); *see also* Ex. 2, FDA, *Constituent Update: FDA Responds to Questions About Levels of Toxic Elements in Baby Food, Following Congressional Report* (Feb. 16, 2021) (Baby food makers "have a legal responsibility … to ensure the safety of their products."). The sale of baby foods in the United States is governed by the Federal Food Drug and Cosmetic Act ("the Act"), 21 U.S.C. §§ 301 *et seq*. Like most foods, baby foods contain several components, including

some from the environment (like lead and arsenic) considered "added" under the Act, and others, which are inherent in the food product and considered "not added" (like caffeine in coffee). *Id.* §342(a)(1). Environmental contaminants, like arsenic and lead, are "added poisonous or deleterious substances." Poisonous or Deleterious Substances in Food, 39 F.R. 42743, *et seq.* (Dec. 6, 1974).

The Act permits the FDA to set tolerance and action levels for harmful substances like heavy metals. *See* 21 C.F.R. §109.4. However, regardless of whether a tolerance or action level is established, it is still the manufacturer's responsibility to reduce the amount of harmful substances in products to as low as reasonably achievable.[3] In 2019 the FDA set an "interim reference level" ("IRL") for lead at 3.0 µg/day. FDA described this level as the level at which lead in food is a concern, noting that no safe level of lead exposure had been identified with respect to neurodevelopment. *See* Ex. 3, Flannery, B., et al. (2020). This IRL, however, did not set a specific limit for the amount of lead that could be present in baby food. In 2020, FDA set an action level for inorganic arsenic in infant rice cereal at 100 ppb. In setting this level, FDA noted that neurodevelopmental toxicity in infants and children was an adverse health effect of concern for inorganic arsenic exposure and that food could be a major contributor. *See* Ex. 4, FDA, *Supporting Doc for Action Level for Inorganic Arsenic Rice Cereal for Infants* (Aug. 1, 2020). No other action levels or tolerances had been set for the products at issue prior to 2021.

In February 2021, the U.S. House of Representatives Committee on Oversight and Reform issued a Staff Report detailing high levels of heavy metals in baby food products sold in the United States. Ex. 5, Staff of H. Comm. on Oversight and Reform, *Baby Foods are Tainted with Dangerous Levels of Arsenic, Lead, Cadmium, and Mercury* (Feb. 4, 2021) ("Staff Report"). The Staff Report revealed internal test results showing high levels of heavy metals in Defendants' baby food products, including levels as high as 641 ppb lead and 180 ppb inorganic arsenic in products and as high as 886.9 ppb lead and 913.4 ppb arsenic in ingredients. *Id*. at 3. For context, if a child consumed a single serving of 100 grams of baby food (infants consume over 1,000g of food per day) at 886.9 ppb of lead, in one serving an infant would consume 88.69 µgs of lead – thirty times the daily IRL

[3] *See, e.g.*, Revisions to the Food Chemicals Codex Policy on Lead and Heavy Metal Specifications, 58 Federal Register 38129 (July 15, 1993).

established by the FDA in 2019 (and forty times the current IRL). If an infant consumed 3 such 100g servings in a single day, it would amount to over 266 µgs of lead. This is why the Staff Report noted that exposure to lead and arsenic "endanger[s] infant neurological development and long-term brain function" and causes "permanent decreases in IQ, diminished future economic productivity, and increased risk of future criminal and antisocial behavior in children." *Id*. at 2. A second Staff Report was released in September 2021 with similar findings from additional baby food makers. Ex. 6, Staff of H. Comm. on Oversight and Reform, *New Disclosures Show Dangerous Levels of Toxic Heavy Metals in Even More Baby Foods* (Sept. 29, 2021).

Regulatory action and legislation followed.[4] The FDA launched the "Closer to Zero" program in November 2021 – a plan focused on determining action levels for lead, arsenic, mercury, and cadmium in foods intended for babies. In announcing Closer to Zero, Dr. Conrad Choiniere, FDA Director of the Office of Analytics and Outreach, explained: "In general the level of each of these contaminants in any single food is low. However overall exposure adds up because many of the foods we eat contain these contaminants in small amounts. This is not to say that we should not be concerned. On the contrary, for the contaminants we are discussing today, we have not identified safe levels of exposure for developmental outcomes." *See* Ex. 8, FDA Tr., *Closer to Zero Action Plan,* at 32 (Nov. 18, 2021). In 2022, FDA lowered the IRL to 2.2 µg of lead per day. *See* Ex. 9, Flannery, B., et al. (2022). According to FDA scientists and other researchers, many children have had exposures that exceed FDA's IRLs. *See* Ex. 10, Spungen, J. (2019); *see also* Ex. 11, Hoffman-Pennesi, D., et al. (2024); Ex. 12, *IFT Comments to FDA on Action Levels for Lead in Food Intended for Babies and Young Children*, at 1 (Mar. 23, 2023). In 2023, FDA issued a final action level for inorganic arsenic in apple juice and a draft guidance for lead in baby food. *See* Ex. 13, FDA, *FDA Action Level for Inorganic Arsenic in Apple Juice: Guidance for Industry* (June 2023); *see also*, Ex. 14, FDA, *Draft Guidance for Industry Action Levels for Lead in Food Intended for Babies and Young Children: Draft Guidance for Industry* (January 2023). In setting the draft action level for lead in baby foods, FDA explained it was "focused on the potential for neurodevelopmental effects

[4] Twenty-three attorneys general also petitioned the FDA to accelerate regulatory actions associated with determining heavy metals action levels. *See* Ex. 7, Ltr. to Food & Drug. Admin. (Oct. 21, 2021).

from lead exposure, as review of the scientific literature indicates that such adverse effects of lead consistently occur at a blood lead level associated with FDA's IRL for children." *Id*. at 5. In January 2025, that draft guidance became final. *See* Ex. 15, FDA Lead Guidance (2025). There, the FDA stated that "[a]lthough no safe level of lead exposure has been identified for children's health, the IRL serves as a useful benchmark in evaluating ***the potential for adverse effects of dietary lead exposure***, such as the ***potential for neurodevelopmental effects***." *Id*. at 6 (emphasis added). The FDA has also announced a target to issue final guidance for arsenic in baby foods and juices in 2025.

While federal efforts to promulgate national standards for levels of heavy metals in baby foods continue,[5] several states, including California, Maryland, Virginia, and Illinois, took action. Each passed laws requiring baby food manufacturers to both regularly test their products and make those results publicly available on the packaging. California's Baby Food Protection Act[6] was the first state law passed.[7] The law requires baby food manufacturers to test finished products and to disclose the results of metal testing online. Legislatures in several states, including Michigan,[8] New York,[9] and Pennsylvania,[10] are currently considering similar bills, with some proposing to lower limits of lead and arsenic in baby foods, citing the risk of neurodevelopmental harms.[11]

II. The Etiology of Autism Spectrum Disorder and Attention-Deficit Hyperactivity Disorder: An Interaction of Genetics and Environment

There is consensus within the scientific community that ASD and ADHD arise because of an interaction between genes and the environment. As a 2019 publication by the National Institute of

[5] *See, e.g*., Baby Food Safety Act of 2021, H.R. 2229, 117th Cong. (2021); Baby Food Safety Act of 2024, S. 4303, 118th Cong. (2023).
[6] Assemb. B. 899, 2023-2024 Reg. Sess. (Cal. 2023).
[7] CAL. DEP'T PUB. HEALTH, *Frequently Asked Questions Assembly Bill (AB) 899: Food Safety-Baby Food* (last updated Jan. 27, 2025). Maryland Illinois, and Virginia's laws mirror California's statute. *See, e.g*., MD. CODE ANN. HEALTH-GEN § 21-330.4 *et seq.*; S.B. 0073, 104th Gen. Assemb., Reg. Sess. (Ill. 2025); H.B. 1844, 2025 Reg. Sess. (Va. 2025).
[8] H.B. 4865, 2025 Leg. 103rd Sess. (Mich. 2025).
[9] A.B. 9026, Gen. Assemb., Reg. Sess. (N.Y. 2025).
[10] H.B. 507, Gen. Assemb., Reg. Sess. (Pa. 2025).
[11] *See* A.B. 9026, Gen. Assemb., Reg. Sess. (N.Y. 2025) ("The legislature hereby finds and declares that toxic heavy metals, including arsenic, cadmium, lead, and mercury, have been detected in baby food products sold in the United States. Even at low levels, exposure to these contaminants may cause significant harm to infants and young children, including impaired neurological development, reduced cognitive ability, and increased risk of developmental and behavioral disorders..."). *See also* H.B. 507, Gen. Assemb., Reg. Sess. (Pa. 2025) (setting limits on the amount of heavy metals in baby food products at 10 ppb for arsenic and 5 ppb for lead and cadmium, and 2 ppb for mercury).

Environmental Health Sciences ("NIH") makes clear, "research shows that both genetics and environmental factors likely play a role in autism spectrum disorder[.]" Ex. 16, NIH, *Autism Spectrum Disorder & The Environment* (Apr. 2019) at 1; *see also* Ex. 17, NIH, Autism at 1 ("A growing area of research focuses on interaction of genetic and environmental factors."); Ex. 18, CDC, *About Attention-Deficit/Hyperactivity Disorder* (Oct. 23, 2024) at 2 ("[S]cientists have identified some possible risk factors" including "[e]xposure to environmental risks" such as "lead during pregnancy or at a young age."). The Centers for Disease Control ("CDC") concurs, stating that "[t]here may be many different factors that make a child more likely to have an ASD, including environmental, biologic and genetic factors."[12] As recently as September 2025, the Society for Developmental and Behavioral Pediatrics ("SDBP") recognized that:

> Decades of research shows that autism has complex causes involving both genetics and environmental influences working together…No single factor—whether genetic or environmental—causes autism on its own. Instead, autism likely results from many different ***genetic variations interacting with environmental factors during critical periods of brain development***.

Ex. 19, SDBP, A Statement from the Society for Developmental and Behavioral Pediatrics on Recent Autism Claims, at 1 (2025) (emphasis added).

To be sure, genes play a role in the disease onset, like they do in any disease. *See* Def. Ex. 16, Shapiro Rpt. at 18-20; Def. Ex. 37, Shapiro Vol. 2 Dep. at 262:12-263:8; Def. Ex. 1, Ritz Rpt. at 38-39; Def. Ex. 4, Hu Rpt. at 21; Def. Ex 17, Reed Rpt. at 19-22; Def. Ex. 10, Aschner Rpt. at 67-68; Def. Ex. 7, Gardener Rpt. at 32-33. For example, genes may explain why, of two children with equivalent heavy metal exposure, only one is diagnosed with ASD. Likewise, environmental exposures may explain why identical twins—who share the same exact genes—do not always share an ASD diagnosis, with 40-80% of twins being discordant for ASD, i.e., only one has been diagnosed with ASD. *See* Def. Ex. 16, Shapiro Rpt. at 18-20; Def. Ex. 4, Hu Rpt. at 21; Def. Ex. 17, Reed Rpt. at 16; Ex. 20, Hallmayer, J., et al. (2011) at 1; Ex. 21, Sandin, S., et al. (2014). For example, a 2011 study at Stanford University School of Medicine found that identical male twins exhibited 42% discordance while fraternal twins exhibited 79% discordance. Ex. 20, Hallmayer, J., et al. (2011) at

[12] CDC, *What is Autism Spectrum Disorder?* (March 25, 2020), *available at*: https://www.cdc.gov/ncbddd/autism/facts.html.

1. This discordance among monozygotic and dizygotic twins shows that genes cannot fully explain ASD risk. If ASD was solely genetic, discordant twins would be exceedingly rare—not 40-80%. *See* Ex. 22, Shapiro Vol. 1 L.R. Dep. at 91:20-92:1; Def. Ex. 13, Guilarte Rpt. at 22-23.

Importantly, it is improper to consider the respective influences of genetics and environmental exposures in terms of strict percentages in any child's risk of developing ASD. ASD is multicausal. Most ASD is explained by a confluence of genetics and environment; when looking at population estimates, *over* 50% could be explained by genetics and *over* 50% could be explained by the environment—not only are they not mutually exclusive, but they often work together. *See* Ex. 23, Shapiro Vol. 1 N.C. Dep. at 213:20-23. Dr. Shapiro explains: "[H]eritability is a population concept. And what it means is the percentage of variance in the symptoms that we observe…in the population that are explained by genetic or environmental factors. It's not a concept that applies to an individual case." *Id*. at 260:11-20; *see also* Ex. 24, Ritz Vol. 1 N.C. Dep. at 194:11-16; Def. Ex. 17, Reed Rpt. at 16. Rather, "in almost every case … there are many contributing factors … that can lead to symptoms, some of which might be genetics, some of which might be environmental exposures." Ex. 23, Shapiro Vol. 1 N.C. Dep. at 82:17-21; *see also* Def. Ex. 36, Shapiro Vol. 1 Dep. at 62:11-63:6 ("[I]t is a combination of various factors that influence the risks and emergence of the symptoms that constitute autism, including genetic factors, environmental exposures and random events that occur in neurodevelopment."); *accord* Def. Ex. 1, Ritz Rpt. at 38; Def. Ex. 17, Reed Rpt. at 16; Def. Ex. 10, Aschner Rpt. at 67. In other words, genes can create susceptibility to ASD that does not manifest into disease absent environmental exposures which, in turn, cause the behavioral and cognitive symptoms diagnosed as ASD. *See* Def. Ex. 36, Shapiro Vol. 1 Dep. at 64:20-65:9; *see also* Ex. 23, Shapiro Vol. 1 N.C. Dep. at 131:18-22, 132:18-133:18, 138:19-24, 141:6-11; Def. Ex. 7, Gardener Rpt. at 62-63. Accordingly, it is inaccurate to assert—as Defendants do—that "genetics (not food) is the primary driver of autism." Def. Br. 3 at 9. Because so is the environment. Def. Ex. 17, Reed Rpt. at 22 ("The proposition that all ASD is only caused [by] genetic factors runs counter to the general scientific consensus regarding the etiology of ASD").

III. A Robust Body of Scientific Evidence Demonstrates that Lead and Arsenic Exposures in Early Life Are Directly Associated with Developing ASD and/or ADHD

The causal link between lead and arsenic exposure and ASD and/or ADHD and their

concomitant symptomology is well established. Unlike other mass torts, where the science is still emerging, here, there is robust agreement and near-consensus within the scientific community that lead and arsenic—including ingested lead and arsenic—cause neurodevelopmental disabilities, which includes ASD and ADHD. There are *hundreds* of epidemiological studies and experiments demonstrating, repeatedly, that children exposed to lead and arsenic in early life sustain brain damage which, in turn, increases the risk of manifesting the behaviors that are diagnosed as ASD and/or ADHD. Whether a specific child's ASD or ADHD is caused by those exposures from baby food is a question of specific causation. For present purposes, Plaintiffs provide a summary of the extensive body of scientific evidence—the same evidence relied on and scrutinized by Plaintiffs' experts—demonstrating that ingested lead and arsenic can interfere with early neurodevelopment and can result in a set of behaviors that can be diagnosed as ASD and/or ADHD.

A. Lead and Arsenic Are Neurotoxic

Lead and arsenic are non-essential heavy metals that provide no benefit to human health—they only cause adverse effects. *See* Def. Ex. 10, Aschner Rpt. at 14-15; Def. Ex. 13, Guilarte Rpt. at 16. One of the most concerning effects of lead and arsenic is on the human nervous system. *See* Ex. 26, ATSDR Lead Profile, at 14 (Aug. 2020) ("Neurological effects of Pb are of greatest concern because effects are observed in infants and children; furthermore, these effects may result in life-long decrements in neurological function."); *see also* Ex. 4, FDA Supporting Document, at 6 ("FDA's risk assessment shows that inorganic arsenic exposure during fetal development, infancy, and childhood may contribute to neurodevelopmental effects[.]"). Once lead and arsenic enter the blood stream, by whatever means, they *cross* the blood–brain barrier ("BBB") by mimicking and utilizing the transporters reserved for beneficial nutrients. *Id.*; Def. Ex. 7, Gardener Rpt. at 52, 92; Def. Ex. 13, Guilarte Rpt. at 33 ("Molecular mimicry of nonessential toxic metals … is defined by their ability to 'hijack' the transporter protein by binding to it and … displacing the essential metal (iron, zinc, copper, and calcium)… resulting in the transport of the toxic metal instead of the essential metal."). Once lead and arsenic enter the brain, they accumulate. *See* Def. Ex. 10, Aschner Rpt. at 14–15. Specifically, the body's system of excreting lead and arsenic from the brain is not as efficient as its mechanisms for uptake and, often, lead and arsenic can bind with macromolecules in the brain,

preventing their excretion entirely. *Id.*

Lead accumulates in those portions of the brain that are known to be affected in children with ASD and ADHD. *See* Def. Ex. 1, Ritz Rpt. at 56–57, 69; Def. Ex. 7, Gardener Rpt. at 71, 74–75; Def. Ex. 10, Aschner Rpt. at 55–57. Lead's neurotoxicity follows multiple pathways, including disruption of energy production through accumulation within mitochondria, inflammation and oxidative stress causing direct neuronal damage, interference with the creation and utilization of neurotransmitters, interference with DNA methylation and non-coding RNAs, and impacting white/grey brain matter development. *See* Def. Ex. 1, Ritz Rpt. at 56–57, 69; Def. Ex. 10, Aschner Rpt. at 29–33; Def. Ex. 4, Hu Rpt. at 18–19, 30–31; Def. Ex. 16, Shapiro Rpt. at 23; *see also* Ex. 26, ATSDR Lead Profile at 262–272 (discussing, in detail, the various mechanisms in which lead interferes with cellular functions). The Cincinnati Lead Study ("CLS") has been exploring the effects of lead on human health, including lead's accumulation in the brain, since the late 1970s. Ex. 28, Cecil, K., et al. (2008) at *0742. The CLS enrolled high-risk mothers and followed them, and their children, for decades, taking regular blood sample s. *Id.* Then, the CLS took MRIs of the adult children's brains. *Id.* at *0742–0743. The data demonstrated that children with higher BLLs between birth and five years old had significantly less grey brain matter development in those portions of the brain that are associated with human behavior and executive functioning. *Id.* at *0744. The CLS researchers explain that these brain matter deficits are "consistent with and potentially explanatory for cognitive and behavioral problems previously associated with lead exposure" which includes "general intellectual and executive functioning, antisocial behaviors, and attention deficit hyperactivity disorder (ADHD)." *Id.* The CLS is one study among hundreds discussed by Plaintiffs' experts, documenting lead's clear and potentially permanent effects on children's brains. *See* Def. Ex. 1, Ritz Rpt. at 53–54.

Arsenic also damages the brain. "Arsenic exposure has been demonstrated to disrupt the formation and modulation of synapses … increase brain inflammation … increase oxidative stress … energy failure, and programmed cell death in the developing brain." Def. Ex. 16, Shapiro Rpt. at 26–27. Arsenic impedes synaptic plasticity of neurons in the hippocampus, interferes with protein creation leading to neurodegeneration, alters the metabolism and utilization of neurotransmitters

interfering with neurodevelopmental pathways, induces oxidative stress, and causes direct neuronal death. *See* Def. Ex. 1, Ritz Rpt. at 62–63; Def. Ex. 10, Aschner Rpt. at 20–21; Def. Ex. 13, Guilarte Rpt. at 27. Multiple studies have reviewed the literature on arsenic and have specifically identified early life exposure to arsenic as a cause of neurodevelopmental harm. *See, e.g.*, Ex. 29, Tolins, M., et al. (2014) at *312 ("[A]rsenic is a developmental neurotoxicant … The neurotoxic effects of arsenic appear to be most severe in the developing brain.").

B. Infants and Young Children Are Uniquely Susceptible to the Neurotoxic Effects of Ingested Lead and Arsenic

The neurotoxic risk posed by exposure to lead and arsenic is compounded by the unique vulnerabilities of infants and young children to toxic heavy metal exposure. Ex. 15, *Action Levels for Lead,* at 4 ("Lead is especially harmful to vulnerable populations, including infants[.]"). Infants and young children consume substantially more food relative to their body weight, as compared to adults, exposing them to higher doses of lead and arsenic from food. *Id.* at 10 ("[C]hildren (0-23 months) … consume larger amounts of food per unit body weight and would therefore have potentially higher exposures."); *see* Def. Ex. 10, Aschner Rpt. at 85-86. Moreover, a child's gastrointestinal track absorbs *more* lead than an adult's, with the ATSDR estimating that children between 2 weeks and 8 years absorb between 40–50% of ingested lead, while adults only absorb 3–10%. Ex. 26, ATSDR Lead Profile at 281; *see* Def. Ex. 1, Ritz Rpt. at 27; Def. Ex. 4, Hu Rpt. at 34; Def. Ex. 7, Gardener Rpt. at 34; Def. Ex. 13, Guilarte Rpt. at 32. Making things worse, infants have a less-developed BBB, which allows lead and arsenic to more easily enter brain tissue and cause damage. *Id.*; *see also* Def. Ex. 10, Aschner Rpt. at 14–15; Def. Ex. 13, Guilarte Rpt. at 32; Def. Ex. 7, Gardener Rpt. at 52. Thus, infants and young children are uniquely susceptible to the risks of toxic heavy metal exposures. Defendants' own expert, Dr. Gabriel Filipelli agrees—in 2010, long before he was retained as Defendants' expert, he published: "[b]ecause of their high absorption efficiency and the rapid neural differentiation during early brain and nervous system development, children are especially vulnerable to permanent effects of lead poisoning[.]" Ex. 30, Filipelli, G., et al. (2010) at 33. Simply put, infants and young children, relative to adults, consume more lead and arsenic in their food, absorb more lead and arsenic from their food, and allow more lead and arsenic to enter their brains.

C. Infancy and Early Childhood Is Characterized by Rapid Brain Development, Which Can be Disrupted by Exposures to Lead and Arsenic

Humans are not born with fully developed brains—"brain development is a continuous process that does not cease at the moment of delivery." Def. Ex. 16, Shapiro Rpt. at 23. After birth, infants experience rapid neurodevelopment. Although the vast majority of neurogenesis, i.e., "the stage at which neurons are generated from precursor cells and migrate to their final destinations in the brain," occurs between the 10th and 25th weeks of gestation, portions of the brain continue to undergo neurogenesis post birth. *Id.* at 15–16. Specifically, postnatal neurogenesis occurs in portions of brain that "are critical for social cognition" and "other cognitive functions," which are often observed as atypical in children with ASD. *Id.*

Humans also undergo synaptogenesis, i.e., the process of forming connections (synapses) between neurons, throughout their life. *Id.* at 16. The most rapid period of synaptogenesis, called exuberant synaptogenesis, occurs between 18 months of gestation and 24 months after birth, reaching its peak at 18 months of age. *Id.* During this period, infants create millions of synaptic connections every day, with substantial synaptogenesis in the prefrontal cortex, "a part of the brain important for attention and executive function as well as social cognition[.]" *Id.*

As exuberant synaptogenesis wanes, around age 2, the process of synaptic pruning accelerates, i.e., the process of eliminating excess synaptic connections. *Id.* Synaptic pruning is an important part of brain development, allowing humans to focus on the most active and relevant neural connections to allow them to efficiently process and respond to stimuli. Indeed, by age 10, 50% of the connections created by synaptogenesis at age 2 will have been eliminated. *Id.* This "balance between synaptogenesis and synaptic pruning in the formation, modulation, and elimination of connections between neurons constitutes synaptic plasticity." *Id.* Research indicates that "altered synaptic stability and plasticity contributes significantly to the emergence of behavioral abnormalities in autism" and that this interference can be caused "by the presence of toxic elements like lead, arsenic, and mercury, which negatively impact synaptic plasticity via mechanisms" described above. *Id.* at 16–17. Specifically, "exposures that disrupt neuronal signaling mechanisms, which are critical drivers of synaptic plasticity" such as lead and arsenic, would be examples of postnatal

environmental exposures that are capable of causing neurodevelopmental deficits.[13] *Id.*

D. A Large Body of Literature Supports the Association of Early Life Exposure to Lead and the Development of ASD

Numerous independent experts—including some of Plaintiffs' experts before being retained here—conclude that early life exposure to lead can cause ASD. For example, in 2016, a consortium of independent scientists, consisting of over 50 of the world's leading epidemiologists, neurologists, toxicologists, health care professionals, and advocates, published a consensus statement in the epidemiological journal Environmental Health Perspectives on behalf of Targeting Environmental Neuro-Developmental Risks ("TENDR"). Ex. 31, TENDR Consensus Stmt. at A118–A122 (2016). The statement was endorsed by numerous academic societies, including the American College of Obstetricians and Gynecologists (ACOG), Child Neurology Society, International Neurotoxicology Association, International Society for Children's Health and the Environment, International Society for Environmental Epidemiology, National Council of Asian Pacific Islander Physicians, National Hispanic Medical Association, and the National Medical Association. *Id.* at A121. The statement was a "a call to action to reduce exposures to toxic chemicals that can contribute to the prevalence of neurodevelopmental disabilities in America's children." *Id.* at A118. They observed that "[w]e are witnessing an alarming increase in learning and behavioral problems in children" which included ASD and ADHD. *Id.* They also "identified 'critical windows of vulnerability'" which included "infancy" and "early childhood" where "toxic chemical exposures may cause lasting harm to the brain that interferes with a child's ability to reach his or her full potential." *Id.* They specifically identified "lead" as an example of a toxic chemical that can "contribute to learning, behavioral, or intellectual impairment, as well as specific neurodevelopmental disorders such as ADHD or autism spectrum disorder." *Id.* at A118–A119.

Numerous other peer-reviewed publications have reached similar conclusions. For example,

[13] It is also worth noting that there is "evidence that abnormal brain metabolism contributes to altered connectivity in individuals with autism." Def. Ex. 16, Shapiro Rpt. at 17. Indeed, a "large number of studies has established that the neurobiology of autism is associated with increased levels of reactive oxygen species, increased lipid peroxidation, and other markers of oxidative stress" and that "individuals with ASD have neurobiological signs of mitochondrial dysfunction … reflecting an alteration in energy metabolism in brain tissue." *Id.* These metabolic issues are some of the exact types of damage caused to neural tissue by exposure to lead. *See* Def. Ex. 1, Ritz Rpt. at 56–57, 69; Def. Ex. 7, Gardener Rpt. at 71, 74–75; Def. Ex. 10, Aschner Rpt. at 55–57.

in a 2020 study looking at metal content in autistic children's hair, researchers concluded that exposures to arsenic and lead, "may play the main role, as an environmental factor, in the pathogenesis of" ASD. Ex. 32, Filon, J., et al. (2020) at 1. In another 2015 study, involving heavy metal exposure and autistic children in Egypt, the authors conclude that "[e]nvironmental exposure to these toxic heavy metals" which includes lead, "at key times in development, may play a causal role in autism." Ex. 33, Mohamed, F., et al. (2015) at 1; *see also* Ex. 34, Goel, A., et al. (2021) at 1 ("[T]he current data and trends suggest a potential strong role for lead in ASD."); Def. Ex. 68, Arora, M., et al. (2017) at 2 ("[E]nvironmental and dietary exposure to metals are potentially important etiological factors in ASD."); Ex. 35, Grandjean, P., et al. (2014) at 1 ("Neurodevelopmental disabilities, including autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive impairments, affect millions of children worldwide, and some diagnoses seem to be increasing in frequency. Industrial chemicals [including lead] that injure the developing brain are among ***the known causes*** for this rise in prevalence.") (emphasis added).

As the Reference Manual notes, "[g]enerally, researchers are conservative when it comes to assessing causal relationships, often calling for stronger evidence and more research before a conclusion of causation is drawn." Ex. 36, Federal Judicial Center, Reference Manual on Sci. Evid. (3d ed. 2011) ("Reference Manual") at 599 (citing *Berry v. CSX Transp., Inc.*, 709 So. 2d 552, 568 n.12 (Fla. Dist. Ct. App. 1998). That multiple independent researchers and renowned scientists are willing to reach causation in published literature is simply a reflection of the strength of the underlying literature linking lead exposure to ASD.

Here, there are over 100 epidemiological studies exploring whether lead exposure is associated with ASD, which includes 16 different systematic reviews and/or meta-analysis. *See, e.g.*, Def. Ex. 4, Hu Rpt. at 22–25. By and large, these studies demonstrate that children with ASD have greater exposures to lead, as established by biomarkers. *See* Def. Ex. 7, Gardener Rpt. at 38 ("A very large number of studies have examined the question of whether a higher body burden of lead during infancy/childhood predicts ASD, and the overwhelming majority of those studies show that lead was measured at higher levels in children with ASD than in children without ASD."); *accord, e.g.,* Def. Ex. 1, Ritz Rpt. at 54; Def. Ex. 13, Guilarte Rpt. at 21.

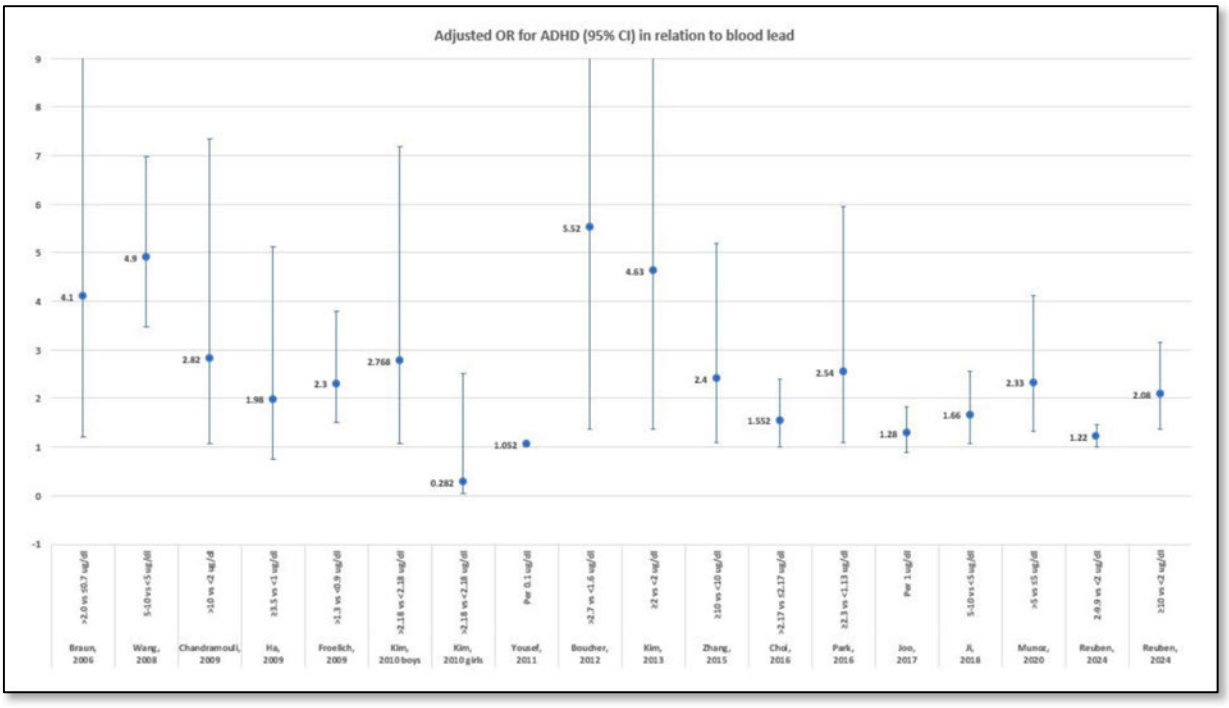
Similarly, the systematic reviews confirm that there is an association between lead exposure and ASD. *See* Def. Ex. 4, Hu Rpt. at 28 ("[T]his collection of systematic reviews and meta-analyses provide a weight of epidemiological evidence in favor of lead exposure having a significant association with ASD, whether viewed in terms of the entire collection of studies, or focusing on the higher-rated studies, or focusing on the more recent reviews and meta-analyses."); *accord* Def. Ex. 13, Guilarte Rpt. at 23–24. Defendants' own expert, Dr. Filippelli, agrees that lead exposure is associated with ASD. Prior to being retained as an expert for Defendants, he published: "[e]ven modestly elevated [blood lead level]'s have been associated with serious neurological disorders such as autism." Ex. 37, Laidlaw, M., et al. (2017) at 10.

Although there are dozens of studies—including various cross-sectional, case-control, and cohort studies—one study deserves special mention. Researchers recruited sets of genetically-identical (monozygotic or "MZ") and fraternal (dizygotic or "DZ") twins and collected sets of each child's baby teeth to test "the hypothesis that prenatal and early life exposure to metal toxicants or deficiency of essential elements during critical developmental windows are associated with ASD.". Def. Ex. 68, Arora, M., et al. (2017) at 2–3. They noted that their "primary focus was on lead, an established neurotoxicant that has been implicated in ASD[.]" *Id.* Within this group, six twin sets (5 MZ and 1 DZ) were concordant for autism (both twins had ASD), seven twin sets (3 MZ and 4 DZ) were discordant (only one twin had ASD), and 19 twin sets (9 MZ and 10 DZ) did not have ASD. *Id.* at 3. Using a laser, the researchers were able to core into each child's teeth and determine, on an incremental basis, how much lead exposure the child had pre and post birth. The researchers found that "[w]hen comparing ASD discordant twins with non-ASD control twin pairs … lead levels were consistently higher in ASD cases than their non-ASD co-twins from 20 weeks before birth to 30 weeks after birth." *Id.* at 4. Lead levels in genetically identical (MZ) discordant twins showed a dramatic difference post birth (starting around 10 weeks postnatal). *Id.* Indeed, "[t]he greatest difference was observed at 15 weeks postnatally, when lead levels in ASD cases were 1.5 times higher … than in their co-twins." *Id.* at 4. The researchers also "compared ASD discordant twin pairs with ASD concordant twins (six pairs) and observed similar patterns … including significantly higher lead levels in cases." *Id.* This study is unique because it was able to measure, within a period

of weeks, a child's postnatal lead levels and see if those differences, when compared to a genetically identical twin's, were associated with ASD. *See* Def. Ex. 1, Ritz Rpt. at 40 ("That is why a study by Arora et al (2017) is particularly notable[.]). Although no single study is dispositive, this study, in conjunction with the large number of observational studies, provides powerful evidence of the effect of early life lead exposure on ASD etiology. *See, e.g.*, *id.* at 45; Def. Ex. 13, Guilarte Rpt. at 22–23; Def. Ex. 7, Gardener Rpt. at 51–52; Def. Ex. 4, Hu Rpt. at 29–30; Def. Ex. 10, Aschner Rpt. at 35.

E. A Large Body of Literature Supports the Association of Early Life Exposure to Lead and the Development of ADHD

The scientific literature demonstrating an association between lead exposure and ADHD is strong. "Out of 27 studies that examined blood lead levels in relation to ADHD, 25 showed that blood lead levels were significantly associated with ADHD." Def. Ex. 7, Gardener Rpt. at 64; *accord* Def. Ex. 1, Ritz Rpt. at 68; Def. Ex. 10, Aschner Rpt. at 48; Def. Ex. 13, Guilarte Rpt. at 25. Dr. Gardener prepared a forest plot to illustrate the results of the 16 studies that reported adjusted odds ratios:



Def. Ex. 7, Gardener Rpt. at 68. Dr. Gardener explains:

> As shown, the 95% confidence bounds excluded 1 (the null) in all but 2 of the studies showing increased blood lead in children with ADHD, indicating that these associations were statistically significant at $p<0.05$. Not only were these results

> highly consistent showing that ADHD is associated with higher blood lead levels, but the graph also displays the substantial strength of the observed associations across studies. In fact, in the majority (11) of the studies the odds ratios surpassed a value of 2.0, indicating that the odds of ADHD were more than double for children above the given blood lead cutpoint.

Id. at 68–69. The data linking BLLs to ADHD strongly supports a causal relationship.

Importantly, the epidemiological literature linking lead exposure to ADHD demonstrates a clear dose response, i.e., greater lead exposure increases risk of ADHD. *See* Def. Ex. 1, Ritz Rpt. at 64–66, 68 ("The effect estimates reported for blood lead and ADHD reflect strong associations with dose response trends."); *accord* Def. Ex. 4, Hu Rpt. at 19; Def. Ex. 7, Gardener Rpt. at 77; Def. Ex. 10, Aschner Rpt. at 48; Def. Ex. 13, Guilarte Rpt. at 25–26. For example, researchers from Johns Hopkins conducted a large prospective birth cohort study with 1,479 mother-infant pairs (299 ADHD, 1180 neurotypical) from the Boston Birth Cohort. Def. Ex. 91, Ji, Y., et al. (2018) at 1–2. The authors "examine[d] the association between early childhood lead exposure and development of ADHD using a prospective birth cohort design." *Id.* at 2. Using BLLs measurements of the children, the researchers were able to follow the cohort to see if anyone developed ADHD and, if so, whether that risk was associated with BLLs. *Id.* at 2–4. They observed an association: "Elevated early childhood blood lead levels increased the risk of ADHD." *Id.* at 1. And, they specifically observed a dose-response relationship: "The natural log-transformed linear trend of lead levels was significantly associated with an increased risk of ADHD diagnosis." *Id.* at 5; *see also* Ex. 38, Geier, D., et al. (2017) at 1 (using FDA data to demonstrate "a significant dose-response relationship between increasing blood Pb levels and the risk of a reported ADD outcome" among U.S. children).

Additionally, there have been 8 systematic reviews and/or meta-analyses of lead exposure and ADHD. Def. Ex. 4, Hu Rpt. at 13–17. Each of the 5 meta-analyses yielded statistically significant positive results. *Id.* And, among the three systematic reviews that did not involve a meta-analysis, the authors noted a clear association between lead exposure and ADHD. *Id.*

F. A Large Body of Literature Supports the Association of Early Life Exposure to Arsenic and the Development of ASD

There is a strong and largely consistent body of literature linking arsenic exposure to ASD: "[T]he current body of epidemiological literature provides substantial support regarding an association between elevated arsenic concentration and increased risk of ASD." Def. Ex. 13, Guilarte

Rpt. at 31; *see also* Def. Ex. 1, Ritz Rpt. at 61; Def. Ex. 7, Gardener Rpt. at 58; Def. Ex. 10, Aschner Rpt. at 26. Although the data is not overwhelmingly positive, like the data involving lead and ASD and ADHD, the epidemiological studies consistently show that ASD children have greater exposures to arsenic than neurotypical children.

For example, a group of independent researchers published a peer-reviewed study looking at the levels of arsenic in the hair of ASD children compared to matched neurotypical children. Ex. 32, Filon, J., et al. (2020) at 1. They surveyed the literature, noting that arsenic is a "well-established neurotoxicants known to cross the blood–brain barrier and affect neurodevelopment Arsenic exposure significantly" and arsenic "affects brain morphology, resulting in gliosis, neuronal degeneration, a decrease in cognitive abilities, attention, comprehension, language skills, and reduces intelligence quotient (IQ) scores[.]" *Id.* at 2. They also noted several other studies demonstrating a link between arsenic exposure and ASD. *Id.* at 3–7. The researchers specifically stated, based on their independent review of the literature, that arsenic "may cause autism" and that "[n]umerous studies have confirmed that heavy metals play a crucial role in the development of autism spectrum disorders." *Id.* at 3, 4. This was confirmed with their own study, which showed that ASD children had four times as much arsenic in their hair compared to neurotypical children. *Id.* at 3. These independent researchers ultimately concluded that arsenic exposure "may play the main role, as an environmental factor, in the pathogenesis of" ASD. *Id.* at 1.

This association is also supported by systematic reviews and meta-analyses. For example, in 2019, researchers reviewed the literature and conducted meta-analyses of arsenic exposure based on data from hair, blood, and urine. *See* Ex. 39, Wang, M., et al. (2019) at *1904. The researchers note that arsenic is "known to cause neurodevelopmental effects when the exposure occurs in early life" and that arsenic has "been proposed to cause neuronal and brain damage that may play a role in the pathogenesis of ASD." *Id.* at *1905. The study, thus, "aimed to systematically review the current literature on human subjects and conduct a metaanalysis to assess the relationship of ASD and iAs[.]" *Id.* Their results demonstrate a consistent association, with every result showing a positive result. *Id.* at *1914. Indeed, the researchers concluded that "[a]fter considering strengths and limitations of the body of research, we concluded that there is consistent evidence supporting a positive association

between early life iAs exposure and diagnosis of ASD[.]". *Id.* at *1904.

This data is confirmed in a more recent meta-analysis. *See* Def. Ex. 64, Ding, M., et al. (2023) at 1–2. The researchers also began with the premise that "[r]ecent studies have shown that the pathogenesis of ASD would be multi-factorial, with genetic, biophysiological, and environmental factors (such as heavy metals exposure) jointly involved." *Id.* at 2. They specified that "[e]nvironmental factors (including neurotoxic heavy metals exposure) play a critical role in the occurrence and progression of ASD." *Id.* In exploring arsenic exposure, they found "that the ASD group had a higher arsenic concentration than the healthy control group ($P < 0.001$)" and that their data "showed that compared with the healthy controls, ASD children had higher arsenic concentrations in their hair, urine, and blood ($P < 0.05$), which further validated the hypothesis that arsenic exposure might be associated with the occurrence of ASD." *Id.* at 10.

Overall, the epidemiological data examining arsenic exposure and ASD is substantial and largely supports an association with ASD.

LEGAL STANDARD

Under Rule 702 and *Daubert*, expert testimony is admissible on three conditions, which courts "liberal[ly]" construe. *Deckers Outdoor Corp. v. Last Brand, Inc.*, No. 23-CV-04850-AMO, 2025 WL 2822682, at *1 (N.D. Cal. Oct. 2, 2025) (quotation marks omitted). First, the proffered expert must be "qualified" to give the testimony by either their "knowledge, skill, experience, training, or education." FED. R. EVID. 702(a). Second, the testimony must be relevant, meaning that the witness's expertise will "help the trier of fact" either "to understand the evidence or to determine a fact in issue." *Id*. Third, the testimony must be reliable, meaning that "the knowledge underlying it has a reliable basis in the knowledge and experience of the relevant discipline." *Decker Outdoor Corp.*, 2025 WL 2822682, at *1 (quotation marks omitted).

The Supreme Court construed these standards in *Daubert v. Merrell Dow Pharm, Inc.*, 509 U.S. 579 (1993), where the Court "firmly rejected" the so-called "'general acceptance' test," under which courts had previously excluded expert opinions based on methodologies that had not "gained general acceptance in the particular field." *Engilis v. Monsanto Co.*, 151 F.4th 1040, 1047 n.6 (9th Cir. 2025) (quotation marks omitted). As the Supreme Court explained, that test was "inconsistent

'with the liberal thrust of the Federal Rules and their general approach of relaxing the traditional barriers to opinion testimony.'" *Id*. at 1047 (quoting *Daubert*, 509 U.S. at 588).

Rule 702 was thereafter amended to include its current "reliability-based requirements," including that "the testimony is based on sufficient facts or data" and that "the testimony is the product of reliable principles and methods[.]" FED. R. EVID. 702(b)–(c); *see also Engilis*, 151 F.4th at 1047–50 (recounting). The *Daubert* Court listed a number of "consideration[s]" that might indicate reliability, such as "whether a theory or technique . . . can be (and has been) tested," whether it "has been subjected to peer review and publication," and, "in the case of a particular scientific technique," "the known or potential rate of error" and "the existence and maintenance of standards." *Daubert*, 509 U.S. at 593–94. These considerations are not requirements; they are not listed in Rule 702, despite several post-*Daubert* amendments. *See, e.g.*, *Engilis*, 151 F.4th at 1050 (applying Rule 702 to a specific-causation opinion without mentioning those considerations); *Mendoza v. Intuitive Surgical, Inc.*, No. 18-CV-06414-LHK, 2020 WL 1976472, at *2 (N.D. Cal. Apr. 24, 2020) ("[T]he Court may permissibly choose not to examine factors that are not 'reasonable measures of reliability in a particular case.") (quotation marks omitted). Nor are those considerations exclusive. *See* FED. R. EVID. 702 (Advisory Committee note to 2000 amendment).

Rule 702 was most recently amended in 2023. This amendment "was 'simply intended to clarify' existing law" in two ways. *Engilis*, 151 F.4th at 1049 (quoting Federal Rule of Evidence 702 (Advisory Committee note to 2023 amendment)). First, the amendment "sought to clarify and emphasize that proffered expert testimony must meet the admissibility requirements of Rule 702 by a preponderance of the evidence." *Id*. (quotation marks omitted). Second, the amendment "emphasize[s] that each expert opinion must stay within the bounds of what can be concluded from a reliable application of the expert's basis and methodology." *Id*. (quotation marks omitted). This emphasis takes the form of a minor word change to Rule 702(d), which now requires, along with the other reliability requirements, that "the ~~expert has reliably applied~~ expert's opinion reflects a reliable application of the principles and methods to the facts of the case." Courts in this circuit—and around the country—have confirmed that this amendment does not change the *Daubert* analysis and have continued to apply Rule 702 the same as before. *See Engilis*, 151 F.4th at 1049 n.9 (noting that "our

decision would be the same under either version of the Rule."). Caselaw from before the amendment accordingly remains relevant.[14] The Advisory Committee notes from the 2023 amendments end with an important caution in considering the 2023 amendments:

> Nothing in the amendment imposes any new, specific procedures. Rather, the amendment is simply intended to clarify that Rule 104(a)'s requirement applies to expert opinions under Rule 702. Similarly, nothing in the amendment requires the court to nitpick an expert's opinion in order to reach a perfect expression of what the basis and methodology can support. The Rule 104(a) standard does not require perfection.

FED. R. EVID. 702 (Advisory Committee note to 2023 amendment).

Indeed, *Daubert* itself "did not work a seachange over federal evidence law." FED. R. EVID. 702 (Advisory Committee note to 2000 amendment) (quotation marks omitted). The Rule's liberal thrust remains, and "the rejection of expert testimony" is "the exception rather than the rule," because "the trial court's role as gatekeeper" under Rule 702 "is not intended to serve as a replacement for the adversary system." *Id*. (quotation marks omitted). As the Ninth Circuit has repeatedly made clear, "the test under *Daubert* is not the correctness of the expert's conclusions but the soundness of his

[14] *See also, e.g.,* Ex. 40, Order re Omnibus *Daubert* Motion, *In re Roundup Prods. Liab. Litig.*, MDL No. 2741 ("The Court's prior *Daubert* rulings do not warrant reconsideration in light of the amendments to Rule 702."); *Hamm v. Mercedes-Benz USA, LLC*, No. 5:16-CV-03370, 2024 WL 3186560, at *1–2 (N.D. Cal. June 26, 2024) (Davila, J.) (rejecting "MBUSA['s] argu[ment] that these changes [to Rule 702(d)] impose a new admissibility requirement"; denying defendant's motion for leave to file a motion for reconsideration because "[t]he Court finds that the changes to 702(d) do not alter the Court's prior conclusion."); *Alivecor, Inc. v. Apple, Inc.*, No. 21-CV-03958, 2024 WL 591864, at *3 (N.D. Cal. Feb. 13, 2024) (White, J.) ("Because the rule change was a clarification, and not a change in the standard, the new language does not materially alter the admissibility of expert testimony.") (citing *Al Qari v. Am. Steamship Co.*, 689 F. Supp. 3d 494, 499 (E.D. Mich. 2023)); *In re NFL's "Sunday Ticket" Antitrust Litig.*, No. ML 15-02668, 2024 WL 2165676, at *3 (C.D. Cal. May 13, 2024) ("Despite the Court having determined that Professor Rascher's testimony was admissible [], Defendants reraise their same objections [] in light of the recent amendments to FRE 702[]. The purpose of the amendments to FRE 702, however, was to clarify – not change – the rule because the Advisory Committee believed that certain courts had misinterpreted the rule by applying an incorrect standard of review. Yet Defendants do not suggest that the Court applied an incorrect standard of review when it denied their motion to exclude Professor Rascher's opinions []. Nor do they point to anything in the Court's previous decision that is inconsistent with FRE 702 as it is written now. As a result, the Court does not find that the amendments to FRE 702 warrant a rehashing of the Court's prior analysis. The Court, therefore, DENIES Defendants' motion."); *McCoy v. DePuy Orthopaedics, Inc.*, No. 22-CV-2075, 2024 WL 1705952, at *9 (S.D. Cal. Apr. 19, 2024) ("Rule 702's 2023 amendments do not represent the sea change Defendants contend. The Advisory Committee wished to correct "certain courts" by clarifying that Rule 104(a)'s preponderance of the evidence standard applies to each of Rule 702's requirements. . . . Defendants give the Court no reason to think the MDL Court misunderstood Rule 702's requirements.[] The Court thus rejects Defendants' argument pertaining to Rule 702's 2023 amendments.").

methodology." *Primiano v. Cook*, 598 F.3d 558, 564 (9th Cir. 2010) (cleaned up). And at the end of the day, "the district judge is a gatekeeper, not a fact finder." *Primiano*, 598 F.3d at 565 (quotation marks omitted). That means "expert testimony does not fail *Daubert* just because a court thinks it is shaky." *In re Accellion, Inc. Data Breach Litig.*, No. 5:21-CV-01155-EJD, 2025 WL 2799102, at *2 (N.D. Cal. Sept. 30, 2025) (quoting *Engilis*, 151 F.4th at 1050). "The evidentiary requirement of reliability" is "lower than the merits standard of correctness." FED. R. EVID. 702 (Advisory Committee note to 2023 amendment) (quotation marks omitted). Thus, even purported instances of "[i]mperfect application of methodology may not render expert testimony unreliable" if the expert still has grounds for his or her conclusions. *Hardeman v. Monsanto Co.*, 997 F.3d 941, 962 (9th Cir. 2021). "When an expert meets the threshold established by Rule 702 as explained in *Daubert*," the necessary result is that "the expert may testify and the jury decides how much weight to give that testimony." *In re Accellion, Inc. Data Breach Litig.*, 2025 WL 2799102, at *2.

ARGUMENT

"In a toxic tort claim for physical injuries," general causation asks whether a plaintiff "was exposed to chemicals that could have caused the physical injuries he complains about." *Engilis*, 151 F.4th at 1045 (quoting *Golden v. CH2M Hill Hanford Grp.*, 528 F.3d 681, 683 (9th Cir. 2008)); *In re Hanford Nuclear Rsrv. Litig.*, 292 F.3d 1124, 1133 (9th Cir. 2002) ("'Generic causation' has typically been understood to mean the capacity of a toxic agent … to cause the illnesses complained of by plaintiffs."); REFERENCE MANUAL at 549 (defining "general causation" as "is the agent capable of causing disease?"). Put another way, general causation asks, "whether exposure to a substance for which a defendant is responsible … is capable of causing a particular injury or condition in the general population." *In re Hanford*, 292 F.3d at 1133. The caselaw and the Reference Manual use phrases like "chemicals," "toxic agent," and "substance for which a defendant is responsible" to describe where, in the causal chain, the general causation question enters. For example, in *Hanford*, the "toxic agent" was ionizing radiation from a nuclear facility owned by the defendant. *Id.* The question was not whether living next to a nuclear powerplant causes an injury; the issue focused on the potential effect of ionizing radiation. *Id.* at 1137. Similarly in *Hardeman*, the "toxic agent" was the chemical glyphosate from a product called Roundup. *See* 997 F.3d at 952. The underlying

studies were not Roundup-specific but instead focused on the chemical or "toxic agent" alleged to cause the harm, i.e., glyphosate. *Id.* at 963 ("To establish general causation, Hardeman's experts needed to show that glyphosate can cause NHL[.]").

In some cases, the "toxic agent" is the same as the source of that exposure. A common example is a typical pharmaceutical case—the drug and the "toxic agent" are often one in the same and, thus, for general causation purposes, the question is whether the product/toxic agent can cause the injury in the general population. In other cases, however, the "toxic agent" is not the same as the source of that exposure, *e.g.*, the radiation and the nuclear powerplant. Indeed, this is common in product liability cases based on a "toxic agent" that is a component of a product, i.e., asbestos in fiberboard, benzene in gasoline, or lead in paint. In those cases, the general causation question does not turn on whether fiberboard, gasoline, or paint cause injury. Instead, the focus is on whether the "toxic agent"—asbestos, benzene, or lead—can cause the injury.

Granted, that does not end the general causation inquiry. As the Ninth Circuit explained in *Hardeman*, general causation requires showing that the "toxic agent" is capable of causing the injury and that it can cause the injury at "exposure levels people realistically may have experienced." *See* 997 F.3d at 963. Thus, Plaintiffs demonstrate that the "toxic substance" can cause an injury and link it realistic, i.e., plausible, levels of exposure. Early on, the Parties discussed the "general causation" question with the Court. *See* Def. Ex. 55, June 20, 2024 CMC Tr. (Dkt. 194) at 33:21–34:10. During that discussion, the Court stated that the general causation inquiry would involve two steps: "The first step is: Can these heavy metals cause these brain abnormalities or dysfunctions that are diagnosed as these … disorders." *Id.* at 33:22–34:2. The second question, is "Do these heavy metals in the defendants' products cause that [injury?]" *Id.* at 34:6–9.[15] Put together, these steps answer the

[15] Plaintiffs' counsel clarified at the hearing what the general causation inquiry would look like:

> We intend to obviously show, based upon the testing, that data we have and that they produce, this is how much metals are in these foods…. And then, you know, for the general causation, our experts are going to look at metals, its effects on the brain, et cetera, and then from there go through the Bradford Hill process and come to a conclusion of whether or not metals can cause it. And then they're going to say, 'Okay, looking at the metals in these foods, would that be capable of causing these levels?' And that's the plan. …. Based on the levels that we see in these foods, it's

question of whether the lead and arsenic in Defendants' baby food products are capable of causing symptoms diagnosed as ASD and/or ADHD. Asked another way, could a child have eaten products from each Defendant with enough lead or arsenic to cause them to experience symptoms that would be diagnosed as ASD or ADHD?

Consistent with the Court's view of the general causation question, Plaintiffs proffer two types of experts. The first are exposure experts Ms. Barr and Dr. Jones, discussed in Part IV, *infra*, who provide an estimate of lead and arsenic exposure that infants could experience consuming specific subsets of each Defendants' baby foods. The second are general causation experts (Drs. Ritz, Hu, Gardener, Guilarte, Aschner, and Shapiro), discussed in Part V *infra*, who address whether ingested lead and arsenic are capable of causing the symptoms that are diagnosed as ASD and/or ADHD, and whether the levels found in Defendants' baby foods could cause and exacerbate those injuries. Together, they provide the basis for general causation.

IV. Plaintiffs' Exposure Experts Offer Admissible Opinions Regarding Plausible Exposures to Lead and Arsenic from Menus of Defendants' Foods (Ms. Barr & Dr. Jones)

A. Plaintiffs' Hypothetical Menus Provide a Plausible Exposure to Heavy Metals from Consumption of Defendants' Baby Foods

General causation is, by definition, not plaintiff-specific. In assessing whether a toxic exposure can have an alleged effect, experts base their opinions on a level of exposure that a theoretical plaintiff might experience—even the highest level. The Hon. Vince Chhabria explained this point in the Roundup MDL: "[T]he inquiry at the general causation phase is not whether" the toxic agent at issue caused the alleged injury in "any of the particular plaintiffs"—plaintiffs "need not establish any particular level of exposure." *In re Roundup Prod. Liab. Litig.*, 390 F. Supp. 3d 1102, 1113 (N.D. Cal. 2018), *aff'd sub nom. Hardeman*, 997 F.3d 941. "It's enough in this litigation, at this stage, for the plaintiffs to show that" the toxic agent "can cause" the alleged injury "when people are exposed to the *highest dose people might plausibly experience*." *Id*. (emphasis added). That

> possible, it's capable of raising the levels in infants to the levels that it would cause the brain damage and lead to the injury.

Def. Ex. 55, June 20, 2024 CMC Tr. (Dkt. 194) at 44:10–45:9. The Court responded: "That's what I had understood. And, yeah, I think they acknowledge that that is what it's going to be." *Id.* at 45:10–11. Defense counsel, Mr. Petrosinelli, agreed, "Right." *Id.* at 45:12.

could include, for example, "a professional gardener who has applied Roundup [the carcinogenic weedkiller] without using protective equipment several times per week, many hours per day, for decades," even if that extreme level of exposure was above what any other plaintiff might have experienced. *Id*. And this makes sense. Defendants are not merely seeking to prevent a specific Plaintiff from getting a jury trial. They are seeking to bar *any* Plaintiff from ever getting a jury trial. The premise of this challenge is that Defendants believe that no Plaintiff will ever be able to prove general causation. If that is so, then Defendants should be able to prevail regardless of any specific Plaintiff, including a plaintiff exposed to "the *highest dose people might plausibly experience*."

In the absence of a specific plaintiff, with specific consumption patterns, Plaintiffs estimated a plausible exposure to lead and arsenic from ingesting Defendants' baby food products in two steps. First, Plaintiffs proffer a plausible set of menus of each Defendant's baby food products and a plausible pattern of consumption of those products during ages when baby food is typically consumed. Second, using Defendants' testing data of those products on the menu, and using the plausible consumption pattern, Plaintiffs have proffered estimates, for each period in the hypothetical child's life, how much arsenic and lead resulted from that consumption.

The first step is done by Plaintiffs' pediatric nutritionist, Pricilla Barr. Ms. Barr established as plausible consumption patterns of baby food for each Defendant. *See* Def. Ex. 20, Barr Rpt., Appendix D (Menus) at 1–34. These menus consist of Defendant-specific baby foods from Appendix A to the Master Complaint. *Id.* They also consider calorie and gram intake information for each product and reasonable estimates about how many servings of baby food a child would consume at different ages. As an example, the Beech-Nut consumption pattern looks as follows:

BEECH-NUT
Consumption Grid

Product	< 1 Month	1- < 4 Mos.	4 - < 6 Mos.	6 Mos. - < 1 yr.	1 - < 2 yrs.	2 - < 3 yrs.
Jars	--	--	3 ser (339g)[1] (135-230 kcals.)	2 ser (226g)[2] (65-170 kcals.)	1 ser (113g) (60-100 kcals.)	--
Pouches	--	--		2 ser. (198g) (105-140 kcals.)	2 ser. (198g) (90-150 kcals.)	2 ser. (198g) (90-150 kcals.)
Cereals	--	--	1 ser (15g) (50 kcals.)	3 ser (45g) (150 kcals.)	3 ser (45g) (150 kcals.)	--
Bars	--	--	--	--	1 ser. (22g) (70-80 kcals.)	2 ser (44g) (150 kcals.)
Yogurt Melts (8+ months)	--	--	--	2 ser. (14g) (50 kcals.)	3 ser. (21g) (75 kcals.)	3 ser. (21g) (75 kcals.)
Baked Crisps	--	--	--	--	--	2 ser. (14g) (50 kcals.)
Subtotal Calories			185-280 kcals.	360-510 kcals.	445-555 kcals.	390-450 kcals.
Total Grams			354g	483g	399g	284g

Assumptions:
1. Did not consume breastmilk.
2. 100% of commercial baby food consumption was Beech-Nut brand.

[1] 1 jar sweet potatoes or carrots (113g – 30-80 kcals.); 2 jars other (apples, green beans, prunes (113g – 35-90 kcals. each))
[2] 1 jar sweet potatoes or sweet carrots (113g – 30-80 kcals.); 1 jar other (green beans; banana, mango & sweet potato; butternut squash & sweet corn; apple, cinnamon & granola; sweet potato & barley (113g 35-100 kcals. each))

Id. at 1. For each Defendant, the menus also list which specific products—for example, which Jars—the hypothetical child consumed within the listed age ranges. *See, e.g.*, *id.* at 2–5 (listing the Beech-Nut products reflected in the Beech-Nut menu). As discussed more fully below, Ms. Barr (an expert nutritionist) reviewed these menus and testified that they reflect diets that a hypothetical child could plausibly consume, i.e., that contain age-appropriate foods with sufficient calories and nutrients.

The second step is taken by Dr. Rachael Jones (an exposure scientist and Professor at UCLA). Dr. Jones calculates how much lead and arsenic a child would consume from these Defendant-specific menus and consumption patterns based on laboratory testing data about these Defendants' products. Thus, in conjunction, Ms. Barr and Dr. Jones provide an estimate of the doses of lead and arsenic that "people might plausibly experience" from eating Defendants' baby food products. *See In re Roundup*, 390 F. Supp. 3d at 1113.

Given that Defendants have dedicated an entire brief to these menus, it is important to put them in proper context. Plaintiffs' menus, along with Ms. Barr and Dr. Jones's testimony about

them, illustrate what the lead and arsenic exposure *could* be for a child that consumed the products on each Defendants' menu. They reflect a possible dose that a child could have realistically experienced, i.e., a plausible dose. At a later date, when this case proceeds to specific causation, the specific consumption pattern and products consumed by the specific plaintiff will be known and used to estimate a lead and arsenic dose for that child. But this testimony is not strictly necessary to general causation. In the *Roundup* litigation, the Court admitted three epidemiology-based opinions "that glyphosate can cause NHL [Non-Hodgkin's lymphoma] at human-relevant doses" without separate expert evidence establishing what a human-relevant dose was. *Id*. at 1151. The Court explained that "there is no need to specify precisely the circumstances under which each plaintiff was exposed to glyphosate" and expressly "acknowledg[ed] that, even at the end of this ruling, precisely what the range of actual human exposure is will remain vague, a product of bifurcated proceedings where the hundreds of individual plaintiffs' experiences remain on the periphery for now." *Id*. at 1115. The Court's concern was ensuring that the plaintiffs' evidence included findings from "doses within the realistic realm of *human* exposure"—what the Court otherwise described as doses that "people might plausibly experience"—and not exclusively findings from laboratory animals. *Id*. at 1113, 1115 (emphasis added). While recognizing that such evidence can itself be reliable and helpful, the Court observed that plaintiffs could not prove general causation based only on "evidence that glyphosate could cause NHL if humans were exposed to glyphosate at the kinds of massive doses, administered in the kinds of ways, that laboratory animals *alone* have experienced." *Id*. at 1113 (emphasis added). Since the plaintiffs' experts did also consider findings from human-relevant doses, however, the Court was able to determine that their opinions were relevant to general causation and sufficiently reliable for *Daubert*. *See id*. at 1127.

The same is true here. Whether lead or arsenic in baby food "can cause" neurodevelopmental injuries "at human-relevant doses" can be seen from the copious human evidence (*e.g.*, epidemiological literature) that Plaintiffs' general causation experts reviewed. *Id*. at 1151; *see infra* Part IV.D-F. Given the depth of this human evidence, there can be no concern that Plaintiffs' general causation experts draw their conclusions based on unrealistically "massive" doses. *Id*. at 1113. Whether Defendants have sold baby foods with at least enough lead and arsenic to "plausibly" expose

a child consumer to the levels reflected in the epidemiological literature—even if only the "highest" hypothetical user—is evident from the testing data available for those foods. *Id*. at 1113. As in *Roundup*, therefore, Plaintiffs can establish general causation based on their experts' opinions that Defendants' products contained enough lead and arsenic, as reflected in the underlying testing data, to cause or exacerbate neurodevelopmental injuries according to the observations in the human evidence. Plaintiffs' general causation experts have accordingly confirmed that they would reach the same conclusions without the Barr or Jones reports. *See* Def. Ex. 26, Ritz Dep. at 295:6-23; *accord* Def. Ex. 35, Guilarte Dep. at 21:10-22:8; Def. Ex. 34, Aschner Dep. at 219:3-220:15; Def. Ex. 29, Hu Dep. at 67:2-5, 199:22-200:11, 314:11-317:14; Def. Ex. 4, Hu Rpt. at 40; Def. Ex. 7, Gardener Rpt. at 90.

All that said, Plaintiffs' menus offer plausible scenarios where each Defendant's products could expose children to dangerous levels of lead and arsenic. The core of Defendants' argument against these menus is that they are not, in their view, "realistic." *See* Defs.' Br. Supp. J. Mot. Excl. Pls.' Exposure Experts Rachael Jones and Priscilla Barr (Dkt. 612) ("Def. Br. 2") at 24–25. However, hypothetical exposures levels are sufficiently "realistic" for general causation purposes if they are within "the highest dose people might plausibly experience." *In re Roundup*, 390 F. Supp. 3d at 1113. And at this stage, Plaintiffs need not offer evidence based on exposures that are identical to what any specific person experienced. *See id*. at 1115. Defendants' more specific attacks are, consequently, irrelevant, though Plaintiffs nonetheless address each herein. It is true that neither Ms. Barr nor Dr. Jones "suggest that the hypothetical meus reflect *typical* consumption patterns for U.S. children." Def. Br. 2 at 24. But "typical" is not the same thing as "plausible." The "highest dose people might plausibly experience" is, by definition, not a 'typical' dose that people experience, and Plaintiffs do not need to present evidence that "typical" U.S. children's consumption patterns of Defendants' baby food products result in lead and arsenic exposures sufficient to cause neurodevelopmental injuries to meet their burden on general causation. Indeed, considering the population of Plaintiffs in this case are a small, self-selected fraction of the population of baby food consumers, it is a group that likely consumed the most contaminated baby foods. Even so, opinions based on any plausible dose, up to and including the highest plausible dose, are relevant and reliable

opinions at the general causation phase. It is also true that neither Ms. Barr nor Dr. Jones claim that the hypothetical menus reflect the "actual, real-world exposures of the plaintiffs in this MDL." Def. Br. 2 at 24. How could they? Knowing what Plaintiffs were exposed to in this MDL would require delving headlong into specific causation—which Defendants have adamantly refused to do. That said, "there is no need" to address that issue now, because it is an issue for specific causation. *In re Roundup*, 390 F. Supp. 3d at 1115. Any distance between the hypothetical menus and any specific Plaintiff's actual exposure is simply "a product of bifurcated proceedings"—on which Defendants insisted — "where the hundreds of individual plaintiffs' experiences remain on the periphery for now." *Id*.

In any event, the hypothetical menus—and resulting exposure levels—are realistic in and of themselves. Defendants' own expert, Dr. Carolyn Scrafford, prepared a datasheet from results of the National Health and Nutrition Examination Survey ("NHANES"). *See* Def. Ex. 25, Gibbons Rpt. at 19, FN 59. Even on that datasheet, there were numerous children who had consumed baby foods in the same categories as the products at issue here at rates equal to or *greater than* those provided in the hypothetical menus. *See* Ex. 42, Barr Vol. 1 Dep., Exhibit 17 (showing the consumptions reported by certain NHANES survey respondents; Ex. 43, Scrafford Vol. 2 Dep. 274:11-275:11, 285:1-8 (explaining that this datasheet relates to the products at issue in the MDL). Defendants' only response is that it is purportedly impossible for a child to consume *these* specific menus, not because it would be biologically or physically impossible for a child to do so, but because the menus supposedly lack variability. *See* Def. Br. 2 at 25. This is the same argument that Dr. Scrafford unreliably makes—and that she should be excluded from making for the reasons in Plaintiffs' Rule 702 briefing. As explained further there, the menus contain caloric ranges that account for daily variability in the specific products that a hypothetical child might eat, and Ms. Barr and Dr. Jones understood the menus that way. *See* Pls.' Mot. to Excl Drs. Scrafford & Gibbons (Dkt. 616) at 16–18. And each menu leaves additional room for variability elsewhere in the child's diet, like table foods. *See infra* Part IV.B.2 (discussing Ms. Barr's conclusions that the menus leave room for additional calorie consumption). Defendants' challenges to admissibility provide no grounds for exclusion.

B. Pricilla Barr Offers Admissible Opinions Concerning the Plausibility of the Hypothetical Menus

1. Ms. Barr Is Highly Qualified

Priscilla Barr is a Neonatal and Pediatric Registered Dietician/Nutritionist with a master's degree in nutrition. Def. Ex. 18, Barr Rpt. at 1. Since completing her Dietetic Internship Training and passing the Registration Examination for Dieticians, she has spent the past 20 years working as a pediatric nutritionist in the Neonatal and Pediatric ICUs at major hospitals in New York City including currently New York Presbyterian Hospital – Weill Cornell Medicine. *Id.*; *see also* Ex. 44, Barr Rpt., Appendix A (Barr CV). In this role, she counsels physicians and families on both inpatient and post-discharge nutrition plans, and helps doctors and parents select diets and dietary plans that incorporate breast milk and/or formula, that transition children to other foods when they are developmentally ready, and that provide the calories and nutrients necessary for healthy development—all experience that is directly applicable to her charge. *See* Def. Ex. 18, Barr Rpt. at 1.

As Defendants do not dispute, Ms. Barr's "knowledge, skill, experience, training, [and] education" qualify her under Rule 702 to render opinions about the plausibility of the hypothetical menus. FED. R. EVID. 702; *see also, e.g.*, *United States v. Pac. Health Corp.*, No. CV1200960RSWLAJWX, 2018 WL 1026361, at *4 (C.D. Cal. Feb. 20, 2018) ("Anyone with relevant expertise enabling him to offer responsible opinion testimony helpful to judge or jury may qualify as an expert witness.") (quotation marks omitted). Indeed, Ms. Barr is the only pediatric nutritionist proffered in this case.

2. Ms. Barr Used A Reliable Methodology to Assess Whether the Hypothetical Menus Were Plausible

Ms. Barr offers two opinions as a pediatric nutritionist: first, that the hypothetical menus "are adequate to meet caloric and key nutrient requirements for each age group of children ages 0–3"; and second, that the hypothetical menus "reflect possible patterns of consumption for American children ages 0–3 during the time period relevant to this litigation" (*i.e.*, 2012–2021). Def. Ex. 18, Barr Rpt. at 3. Both of these opinions reflect a rigorous and reliable application of Ms. Barr's knowledge and experience to the types of materials and methods that she uses in her everyday work.

To reach her first conclusion—on caloric and nutritional adequacy—Ms. Barr calculated the

caloric needs for each age range in the hypothetical menus based on the Dietary Reference Intake Estimated Energy Requirements ("EER"). *See* Def. Ex. 18, Barr Rpt. at 7. These are part of the Dietary Reference Intakes developed by the Food and Nutrition Board of the National Academies of Sciences, Engineering, and Medicine, and they are the recommended equations for calculating caloric requirements. *See id*. at 7; *see also* Ex. 45, National Academies of Sciences, Engineering, and Medicine (2023) at 11-13. Per the CDC's recommendation, Ms. Barr drew the heights and weights for these equations from the World Health Organization ("WHO") Growth Charts for the 0–2 age range and from the CDC's growth charts for the 2–3 age range. *See* Def. Ex. 18, Barr Rpt. at 7; *see also* Ex. 46, Grummer-Strawn, L., et al. (2010) at 2 ("The WHO charts are growth standards that describe how healthy children should grow under optimal environmental and health conditions."). To assess whether the hypothetical menus would be adequate for most children, Ms. Barr assessed whether they would provide sufficient calories for children at the 3rd and 97th percentiles of the WHO growth charts and at the 5th and 95th percentiles of the CDC growth charts. *See* Def. Ex. 18, Barr Rpt. at 7–8. That process yielded estimated average energy (*i.e.*, calorie) needs for children at the high and low height/weight ends of each age group. *Id*. at 9 tbl. 1 (EER calculations).

Ms. Barr then assessed whether the hypothetical menus of Defendants' baby foods would provide adequate calories for each of these groups in the context of overall diets that also include infant formula, cow's milk, and table foods. To do so, she estimated the number of calories that children in age ranges from 6 months to 3 years would consume from table foods from a study based on data in the 2002 Feeding Infants and Toddlers Study ("FITS"), conducted by Defendant Gerber. *See id*. at 9–10. Again, to account for a broad swath of potential children, she used the table-food consumption estimates for the 25th percentile of this data, which enabled her to assess whether the hypothetical menus of Defendants' baby food products could provide necessary calories to those children who eat relatively fewer table foods. *See id*. at 10. She also estimated the number of calories that children would otherwise receive from infant formula or cow's milk based on the sources and recommendations about age-based dietary habits thoroughly reviewed earlier in her report. *See id*. at 3–7, 10. For each age group (6 months to <1 year, 1 to <2 years, and 2 to <3 years), Ms. Barr ultimately found that the hypothetical menus of each Defendant's commercial baby foods

would provide the remaining calories that children even at the 97th WHO or 95th CDC height/weight percentiles would need after consuming the estimated amounts of infant formula, cow's milk, and table foods. *See id*. at 11. In other words, each of these menus could plausible "provide sufficient calories from baby food to sustain the overwhelming majority of children ages 0–3." *Id*. Ms. Barr also determined that these menus would provide adequate iron and other essential nutrients (including calcium, folate, zinc, and protein) based on the relevant products' labels and on the various nutritional guidelines that she commonly applies. *See id*. at 3–7, 12–13.

To reach her second conclusion—that the consumption patterns reflected in the hypothetical menus (*e.g.*, one daily serving of Beech-Nut jars for 1-year-olds) are plausible—Ms. Barr considered children's relative developmental feeding abilities as well as national data about children's actual caloric intake, consumption of commercial baby foods, and snacking habits based on a review of the relevant literature. *See id*. at 13–25; *see also* Ex. 47, Barr Rpt. Appendix C (Barr MCL). Based on relevant product labels, dietary recommendations from the American Academy of Pediatrics and others, and data from the 2016 FITS as well as NHANES, among other sources, Ms. Barr concluded that the hypothetical menus "reflect realistic ages of introduction and consumption for the food products." *See* Def. Ex. 18, Barr Rpt. at 14–17.

As for intake, Ms. Barr assessed not only whether the hypothetical menus would provide enough calories (per the above), but also whether those calories would exceed the actual intakes observed in national data. *See id*. at 18. To do this, she again relied on data from FITS, which was generally consistent with data from NHANES. *See id*. And, she again relied on a relatively high percentile (in this case, the 90th percentile average intake observed in the data) to ensure that her estimates accounted for a broad swath of children, i.e., to calculate estimates of caloric intake that "most, but not all, same-aged children's caloric intakes would fall below." *Id*. According to the calories listed on Defendants' product labels, Ms. Barr concluded that none of the consumption patterns reflected in the hypothetical menus would—even in conjunction with previously estimated formula and milk consumption—exceed the 90th percentile of caloric intake reported in FITS. *See id*. at 19. These menus generally also left substantial room for additional consumption of table foods. *See id*. at 19–20. Thus, Ms. Barr concluded, "[t]here are multiple plausible scenarios in which a child

could consume Defendants' baby food products as laid out in [the hypothetical menus] without exceeding 90th percentile intake levels." *Id*. at 20. A child aged 6 months to 1 year "could follow these consumption patterns of Defendants' baby food products and receive up to 12–29% of his or her energy from table foods (depending on the specific menu)" without exceeding that level. *Id*. Children aged 1 to 2 years could likewise consume a diet of 37–59% table foods, and children aged 2 to 3 years could consume up to 45–69% table foods. *See id*.

Finally, Ms. Barr concluded—again based on data from FITS and NHANES as well as the FDA—that the hypothetical menus are plausible in light of U.S. children's actual observed consumption of commercial baby foods and snacking habits. *See id*. at 21–25. For example, the average consumption of dry infant cereal across the menus was within less than half a teaspoon of the 90th percentile intake measured by the FDA. *See id*. at 22. Consumption of single-ingredient root vegetable products was also generally consistent with national data. *See id*. at 24. And although Ms. Barr found that the hypothetical consumption rates were in some instances higher—on a grams-per-day basis—than the observed averages or 90th-percentile measurements for certain products on certain menus (namely, certain baby food and fruit mixtures), this finding did not alter her conclusion "because the caloric intake from those products is comparable to the caloric intake from other Defendants'" comparable products. *Id*. at 23. In "ordinary practice," consumption patterns are discussed in terms of calories because they are "more indicative of nutritional content." *Id*.

All these measurements rely on sources and methods that Ms. Barr uses in her daily practice advising doctors and patients about healthy and adequate diets for young children. *See* Def. Ex. 39, Barr Vol. 1 Dep, at 33:4-16; 37:4-15; 40:4-41:58; 311:17-23 (explaining her background familiarity with NHANES, FITS, and nutritional studies involving statistics and epidemiology). Throughout, Ms. Barr was careful to recognize the limits of her analysis. *See* Def. Ex. 18, Barr Rpt. at 11, 17, 20-21, 24-25 (noting that these are just averages and that specific children will differ). As a result, she can reliably testify that Plaintiffs' hypothetical menus are plausible along the vectors she considers, namely nutritional adequacy and consistency with actual consumption patterns. And, at the general causation phase, that testimony will help the factfinder understand why these menus reflect exposures that children "might plausibly experience." *In re Roundup*, 390 F. Supp. 3d at 1113.

3. Defendants' Challenges to Ms. Barr's Opinions Are Unavailing

Defendants do not contest the reliability of any of the methodology described just above. In their motion to exclude Ms. Barr, Defendants do not mention EERs, FITS, NHANES, or any of the underlying published methods and data she relied on—let alone contest that these methods and sources provide a reliable basis for the type of analysis she conducted. Nor do they develop any argument as to how Ms. Barr might have failed to reliably employ those sources and methods in reaching her conclusions. There is thus no actual dispute that Ms. Barr's analysis reflects reliable methods reliably applied, as Rule 702 requires. And, Defendants do not dispute the relevance of that analysis, that is, of the factfinder's ability to understand that the hypothetical menus, which Plaintiffs offer as examples of plausible exposure scenarios, are indeed plausible from a dietary perspective.

Defendants instead offer two general arguments for excluding Ms. Barr. First, they contest the concept of assessing hypothetical menus at all. They recognize, however, that experts are allowed to—and often do—rely on attorney-provided facts and assumptions. Indeed, this is why the Federal Rules require experts to "identify facts or data that the party's attorney provided and that the expert considered in forming the opinions to be expressed" and any "assumptions that the party's attorney provided and that the expert relied on in forming the opinions to be expressed." FED. R. CIV. P. 26(b)(4)(C)(ii)–(iii). That an expert's testimony might be based on such facts or assumptions "is not a bar to her testimony." *United States ex rel. Jordan v. Northrop Grumman Corp.*, No. CV 95-2985 ABC (EX), 2003 WL 27366224, at *6 (C.D. Cal. Jan. 6, 2003) (citing *United States v. Soulard*, 730 F.2d 1292, 1299 (9th Cir. 1984)); *accord Miguel v. Salesforce.com, Inc.*, No. 20-CV-01753-MMC, 2024 WL 1221934, at *3 (N.D. Cal. Mar. 20, 2024). As always, the question under *Daubert* is whether an expert applied reliable methods to those facts and assumptions in reaching his or her conclusions. *See, e.g.*, *Sardis v. Overhead Door Corp.*, 10 F.4th 268, 281 (4th Cir. 2021) ("[T]o the extent an expert makes inferences based on the facts presented to him, the court must ensure that those inferences were 'derived using scientific or other valid methods.'"); *see also infra* Part IV.C.3 (citing further cases in response to same challenge to Dr. Jones).

To be sure, expert testimony may be excludable where "the indisputable record contradicts or otherwise renders the opinions unreasonable," *De la Torre v. CashCall, Inc.*, 56 F. Supp. 3d 1073,

1095–96 (N.D. Cal. 2014), or, similarly, where the testimony is "based on assumptions that are so unrealistic and contradictory as to suggest bad faith or to be in essence an apples and oranges comparison," *Zerega Ave. Realty Corp. v. Hornbeck Offshore Transp., LLC*, 571 F.3d 206, 214 (2d Cir. 2009) (quotation marks omitted). But this is far from such a case. For each hypothetical menu, Ms. Barr was asked to make two assumptions: first, that a hypothetical child consumed commercial baby food (in addition to milk/formula and table food) from only that particular Defendant, thereby enabling her to assess, pursuant to "step two" of the general causation analysis that this Court articulated, the baby food exposures that a consumer might plausibly experience from that Defendant; and second, that the child consumed only infant formula rather than breastmilk, which is a realistic assumption[16] for the reasons in her report. *See* Def. Ex. 18, Barr Rpt. at 4. Otherwise, her analysis of the menus relies (in addition to all the published methods and data she cites) on the caloric content and serving sizes of the relevant products—which are facts provided by Defendants themselves, and which she confirmed based on product labels. *See id*. at 15. There is thus "a basis in the record supporting [Ms. Barr's] factual assumptions." *Sloan v. Gen. Motors LLC*, No. 16-CV-07244-EMC, 2020 WL 1955643, at *38 (N.D. Cal. Apr. 23, 2020) (quotation marks omitted). That is all that is needed for her to reliably analyze whether the proposed menus provide adequate nutrition and fit national consumption data, i.e., determine whether they are plausible. And again, Defendants do not challenge the reliability of the actual steps of that analysis, which is ultimately what matters under *Daubert*. Any further attack on the underlying facts or data "are issues that go to impeachment and weight, not admissibility." *Id*. (noting that "it cannot be said that [the expert's] opinion is not based on facts or data" where, "to the contrary, it is based on data provided to him by Defendant."); *see also In re Tesla, Inc. Sec. Litig.*, No. 18-CV-04865-EMC, 2022 WL 7374936, at *11 (N.D. Cal. Oct. 13, 2022) (noting "in general such issues are for the jury to assess" as long as "there is a basis in the record supporting" the challenged assumption).

The supposedly "towering series of unsupported . . . assumptions" that Defendants attempt to erect crumbles in the face of Ms. Barr's actual analysis. Def. Br. 2 at 13. As Defendants point out,

[16] Many mothers can struggle with breastfeeding. And, as acknowledged by Defense expert, Dr. Scrafford, 56% of infants 4 - <6 months are "non-nursing"—a percentage which only grows by age bracket (climbing to 92% non-nursing infants 1 year - <2 years, etc.). Ex. 48, Scrafford Rpt. at 35.

Ms. Barr did not select the particular products or consumption patterns reflected in the hypothetical menus. But that would be the case in any non-bifurcated case, where the products consumed by the child would be dictated by the specific facts of that child's case, not selected by an expert. Indeed, this is simply another way of describing the premise of her review, which was whether the hypothetical menus and consumption patterns were plausible from a nutritional and consumption standpoint. Ms. Barr also did not "compare the products on the hypothetical menus to the list of products at issue in the MDL" or "make any affirmative suggestions to specific products or quantities to include in the hypothetical menus." *Id*. at 15. But Defendants make no effort to explain why either of those steps would be relevant to—let alone necessary for the reliability of—the analysis she did conduct. Moreover, as Defendants themselves note, Ms. Barr rejected certain initial menus that she found facially implausible based on the standards laid out in her report. *See id*. at 6; Def. Ex. 39, Barr Dep. Vol. 1 at 114:2-115:21. All of this reflects a rigorous application of a reliable methodology to the facts of the case.

To be clear, Ms. Barr did not "blindly" rely, as Defendants repeatedly assert, on anything that Plaintiffs' attorneys provided, she formed opinions about the contents of the hypothetical menus based on her own independent application of methods and materials common to her daily practice, as thoroughly explained in her report. Ms. Barr did not need to participate in the formation of the assumptions that she was given to reliably opine about whether those assumptions are plausible. That is the nature of testifying about given assumptions, which experts may and routinely do. Defendants certainly remain free to point out to the factfinder that the hypothetical menus do not include all the products at issue in the MDL—but, so what? If any subset of Defendants' baby food products could cause the injuries at issue, general causation is established. Defendants' quibbles with the menus have nothing to do with the reliability of Ms. Barr's own independent assessment of those menus. After all, "experts routinely base their opinions on assumptions that are necessarily at odds with their adversary's view of the evidence." *Artunduaga v. Univ. of Chicago Med. Ctr.*, No. 12 C 8733, 2016 WL 7384432, at *5 (N.D. Ill. Dec. 21, 2016) (cleaned up). That is not an issue under Rule 702.

Second, Defendants attack certain "assumptions" that purportedly underlie Ms. Barr's opinions about the hypothetical menus' plausibility. Def. Br. 2 at 21. These attacks ultimately

amount to an argument that the menus are not realistic. And, as already explained, that argument fails. *See supra* Part IV.A. Defendants specifically fault Ms. Barr for failing to somehow "validate" the hypothetical scenarios reflected in the menus for each Defendant. Def. Br. 2 at 22. But this is not an attack on methodology, because Defendants do not identify anything else that Ms. Barr should have or failed to do. Indeed, it is not clear what such a validation would constitute absent a wholesale analysis of case-specific facts of Plaintiffs in this MDL—a task that clearly falls outside of general causation. This argument is, instead, simply an observation that the menus depict a potentially limited class of consumers (i.e., ones that eat a range of particular product types from a particular Defendant). Again, this is not an attack on whether the opinions Ms. Barr provides are reliable under Rule 702—it is simply an attack about what her otherwise admissible opinions mean.

Defendants similarly argue that the menus include children in some age ranges eating foods meant for younger children. This, again, is not an argument about whether this consumption is plausible. *See In re Roundup*, 390 F. Supp. 3d at 1113. Older children eat foods designed for younger children all the time, especially if that child really likes a particular product. Defendants own two examples illustrate this point. Defendants point to Beech-Nut's "Rice Cereal" product, which they call "infant" rice cereal and claim, without any specific support, that children would not consume rice cereal up to 2 years of age. This is contrary to the peer-reviewed literature cited by Ms. Barr demonstrating that some children do continue to consume infant cereal through age 2. *See* Def. Ex. 18, Barr Rpt. at 21. Defendants also reference Nurture's Pea & Spinach Teethers, which they claim children also would not consume up to 2 years of age (even though children may still be teething). As Ms. Barr notes, however, these products indicate no maximum ages. *See* Def. Ex. 20, Barr Rpt. Appendix D (Menus), at 5, 18. And, Ms. Barr also cites literature indicating that many children continue to consume "infant finger foods" like teethers through age 2. *See* Def. Ex. 18, Barr Rpt. at 21. This is a reliable basis to support her conclusion as to the plausibility of the menus under Rule 702 on this point.

Finally, Defendants argue that some hypothetical menus contain products that were not available during the entirety of the designated consumption periods. But that limitation was indicated *on the menus themselves*. *Compare* Def. Br. 2 at 23 (noting that the two Bowls on the Nurture menu

"only came onto the market sometime in 2018"), *with* Def. Ex. 20, Barr Rpt. Appendix D (Menus), *accord* at 18 & nn.29–30. Defendants thus fail to find any legitimate fault with Ms. Barr's analysis of the hypothetical menus. Her testimony is relevant, reliable, and admissible under Rule 702.

4. Dr. Rachael Jones Offers Admissible Opinions Concerning the Estimated Lead and Arsenic Exposure from the Hypothetical Menus

a. Dr. Jones Is Highly Qualified

Dr. Rachael Jones, PhD, MPH, CIH is a Professor and Chairwoman of the Department of Environmental Health Sciences at the University of California, Los Angeles (UCLA) Fielding School of Public Health. *See* Def. Ex. 21, Jones Rpt. at 6. She also directs the UCLA Center for Occupational and Environmental Health, a state-funded research and educational center that provides outreach and technical support to advance occupational and environmental health in California. *Id*. Dr. Jones received both a Master of Public Health degree with emphasis in industrial hygiene and a Doctor of Philosophy degree in environmental health sciences from the University of California, Berkeley. *See id*. Dr. Jones completed post-doctoral training in interdisciplinary public health at the University of Illinois, Chicago. *See id*. Dr. Jones is a Certified Industrial Hygienist which is a specialty in exposure science focused on the assessment and control of exposures. *See id*. She has secured millions of dollars in grants and contracts to conduct exposure assessment and exposure control research, including from the Agency for Health Care Research and Quality, the CDC, the Department of Defense, the National Institute of Allergy and Infectious Diseases and the University of California Office of the President. *See* Ex. 49, Jones Rpt. Appendix A (Jones CV). Dr. Jones has published approximately 95 articles in the peer-reviewed literature, most of which are related to methodological development and/or application of exposure science methods to real-world problems. *Id*. She currently serves as Chief Editor of Annals of Work Exposures and Health, the journal of the British Occupational Hygiene Society published by Oxford University Press. *See* Def. Ex. 21, Jones Rpt. at 6.

5. Dr. Jones Used a Reliable Methodology to Estimate Lead and Arsenic Exposure from Consumption of the Products on the Hypothetical Menus

Dr. Jones reliably calculated the exposures to lead and arsenic that would result from consumption of menus of each of the Defendants' baby foods, consistent with each validated

consumption pattern. The "hypothetical" menus set forth an assortment of products eaten per category and an average consumption amount of each product category over different time periods in a child's development through 3 years. *See* Def. Ex. 20, Barr Rpt., Appendix D (Menus); *see also* Def. Ex. 21, Jones Rpt. at 10; Def. Ex. 41, Jones Vol. 1 Dep. at 159:9-23; Def. Ex. 42, Jones Vol. 2 Dep. at 510:13-24; Def. Ex. 40, Barr Vol. 2 Dep. at 435:15-437:4. Calculating exposures based on a "hypothetical" menu is not a fundamentally different exercise than calculating exposure for a specific plaintiff based on plaintiff-provided information—something courts in this circuit routinely admit expert testimony about. *See e.g.*, *In re Roundup Prods. Liab. Litig.*, 732 F. Supp. 3d 1091, 1093–94 (N.D. Cal. 2024) (admitting expert testimony on the Plaintiff's exposure to Roundup where that exposure calculation was based on information in plaintiff fact sheets and plaintiffs' deposition testimony); *see also Exxon Mobil Corp. v. AECOM Energy & Constr., Inc.*, No. CV 19-107-BLG-SPW, 2024 WL 5245649, at *3 (D. Mont. Dec. 11, 2024) ("An expert is permitted to testify to an opinion formed on the basis of information that is handed to them rather than developed by them, as long as the court makes sure that the expert isn't being used as a vehicle for circumventing the rules of evidence.") (internal citation omitted).

To quantify exposures to lead and arsenic that would result from consumption of these menus, Dr. Jones used a scenario evaluation or "reconstruction" approach, which is an approach endorsed by the U.S. Environmental Protection Agency ("EPA"). *See* Def. Ex. 21, Jones Rpt. at 7-8; Ex. 50, EPA, *Exposure Factors Handbook: 2011 Edition* at 1-14 (Sept. 2011). This approach is consistent with the exposure assessment process as outlined in the Reference Manual. *See* Ex. 36, Reference Manual at 507 ("Exposure assessments can be directed at past, present, or even future exposures and can be narrowly focused (one chemical, one environmental medium, one population group)…"). Dr. Jones utilized recognized exposure equations to calculate average daily dietary exposures for each life stage consistent with guidance from EPA. *See* Def. Ex. 21, Jones Rpt., at 9-11; *see also* Ex. 36, Reference Manual, at 525; Ex. 51, Scrafford Vol. 2 Dep, Exhibit 41 at 11 (PowerPoint authored by defense expert Dr. Scrafford endorsing same basic exposure equation utilized by Dr. Jones); Ex. 52, EPA, G*uidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants* (2005).

To determine lead and arsenic concentration for each product, Dr. Jones used data experts in her field typically rely on—analytical laboratory testing data. Specifically, Dr. Jones relied on heavy metal testing data produced by Defendants along with heavy metal testing conducted by third parties. *See* Def. Ex. 21, Jones Rpt. at 5. Defendants' testing data was compiled from documents identified as containing unique and relevant test results by Defendants in interrogatory responses. *See e.g.*, Ex. 53, Beech-Nut Nutrition Company's First Supp. Responses and Objections to Plaintiffs' First Set of Interrogatories (May 12, 2025) at 18-22, 29-32. Relying on exposure inputs that originate from information provided by parties' sworn testimony is entirely appropriate. *See, e.g.*, *In re Roundup*, 732 F. Supp. 3d at 1095. Consistent with her training and professional experience, she conducted her data analysis utilizing the R Project for Statistical Computing Software. *See* Def. Ex. 21, Jones Rpt. at 13. Her analysis of the data included creating unique product identifiers to ensure proper linking between ingredient and finished product test results to the products used in her calculations. *See id*. at 12-13. She endeavored to exclude all duplicates and tests reflecting rejected product or ingredient lots—relying primarily on the identification results listed in Defendants' sworn discovery responses—and cleaned and verified the data as laid out in her reports. *See id*. at 12-13; *see also* Def. Ex. 24, Jones Amended Rebuttal Rpt. at 2; Def. Ex. 41, Jones Vol. 1 Dep. at 19:9-20, 24:5-14.

For each finished product on the menu, Dr. Jones calculated a mean and maximum arsenic and lead concentration. *See* Def. Ex. 21, Jones Rpt. at 10. As described in her report, her calculations used finished product testing, where available, and ingredient testing and product formulas where there was limited or no finished product testing available. *See id*. at 9-10, 12. Because there was insufficient testing data to calculate inorganic arsenic concentrations for the products, Dr. Jones used the overall arsenic levels reported in the testing data to calculate total arsenic ingestion. *See* Def. Ex. 21, Jones Rpt. at 10; *see also* Def. Ex. 24, Jones Amended Rebuttal Rpt. at 32. However, where available, Dr. Jones summarized inorganic arsenic testing data and described the percentage of arsenic comprised of inorganic arsenic in Defendants' baby food products. *See* Def. Ex. 21, Jones Rpt. at 5; *see also* Def. Ex. 24, Jones Amended Rebuttal Rpt. at 32. The mean and maximum intake rates of lead and arsenic, based on the validated consumption patterns, were summed across products and product types to calculate a typical (mean) and worst case

(maximum[17]) daily intake of lead and arsenic expected to result from consumption of products in each Defendant menu, for each life stage. *See* Def. Ex. 21, Jones Rpt. at 5, 10.

Dr. Jones also used the IEUBK Model developed by the EPA to calculate the geometric blood lead level ("BLL") expected to occur as a result of the average daily intake of lead from each Defendants' products. *See* Def. Ex. 21, Jones Rpt. at 5; *see also* Def. Ex. 24, Jones Amended Rebuttal Rpt. at 32. The IEUBK model is a reliable method. *See Blanks v. Fluor Corp.*, 450 S.W.3d 308, 370 (Mo. App. 2014) (upholding on appeal a jury verdict where causation was supported by a toxicologist's estimation of the plaintiffs' blood lead levels using the IEUBK model); *see also A.O.A. v. Rennert*, No. 4:11 CV 44 CDP, 2025 WL 1918941, at *8 (E.D. Mo. July 11, 2025) (allowing competing testimony about the superiority of single vs. multi-factorial models to predict BLLs). The IEUBK model has been extensively studied and "was designed to 'provide appropriate, unbiased, estimates of blood lead concentrations' owing to model parameter values being selected to be 'reasonable best estimates,' rather than 'building conservatism' into the model predictions…" Def. Ex. 21, Jones Rpt. at 10 (quoting Ex. 54, White P.D., et al. (1998) at *1526). The IEUBK model calculates a geometric mean BLL for a hypothetical child when a single run is performed using specific inputs. *See* Def. Ex. 21, Jones Rpt. at 10; *see also* Ex. 55, SRC Inc., *User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) version 2.0,* at 13 (2021). To isolate the BLL contribution from consumption of baby food alone, Dr. Jones only calculated values for dietary lead exposure from the menus as the dietary intake input. *See* Def. Ex. 21, Jones Rpt. at 11. This meant setting the other input exposures (air, soil, water, etc.) to zero, which does not affect the reliability of the outputs and allows the IEUBK model to isolate the contribution to BLL from the baby food products.[18] *See id.*

In response to Defense expert reports and new information— documents not produced prior to her report submission date and documents not identified as duplicates or rejected ingredients in

[17] Maximum value calculations were selected to represent the worst-case scenario in which the hypothetical infant or young child ate only the products with the highest lead and arsenic concentrations. *See* Def. Ex. 21, Jones Rpt. at 9.

[18] It is well-settled in this circuit that "'[a] slight modification of an otherwise reliable method' does not render expert testimony inadmissible." *Hardeman*, 997 F.3d at 961–62 (quotation marks omitted). This is especially so considering it makes no mathematical difference here.

response to interrogatories—Dr. Jones revised her data set and updated her calculations on July 15, 2025, and again on August 29, 2025.[19] Dr. Jones's methodology was consistent across her reports. *See* Def. Ex. 23, Jones Rebuttal Rpt. at 6; Def. Ex. 24, Jones Amended Rebuttal Rpt. at 2. Dr. Jones's calculations of mean and maximum daily intake of lead and arsenic and blood lead levels for each life stage for each of the provided menus are summarized on page 33 of her Amended Rebuttal Report. *See id*. She used validated menus and consumption patterns, Defendants' own data and discovery responses, and standard exposure methods to arrive at her results.

6. Defendants' Challenges to Dr. Jones's Opinions Are Unavailing

Defendants do not, nor could they credibly, argue that Dr. Jones is unqualified to render these opinions. Defendants also do not attack the reliability of her methods used to calculate exposures. They do not criticize her use of Defendants' internal or third-party data or the calculations she used for external dose, her use of the IEUBK model to calculate an internal blood lead level, or the method she selected for dealing with left-censored data. Instead, Defendants offer several criticisms related to the facts that Dr. Jones calculated an exposure based on menus provided by Plaintiffs' counsel, and validated by Ms. Barr, and that there were initial (now corrected) errors in her dataset that led her to revise her calculations (the accuracy of which they do not challenge). These arguments amount to attacks on the limitations of the scope of Dr. Jones's opinions or weak grist for cross examination. They are not grounds for exclusion.

a. Dr. Jones Exercised the Level of Intellectual Rigor of Experts in Her Field in Calculating Exposures Based on Validated Consumption Patterns and Proposed Menus

Contrary to Defendants' unsupported assertion, there is no mandate that a scientifically valid exercise in exposure science requires population-level modeling of "typical" ingestion data. Def. Br. 2 at 14. Indeed, the Reference Manual makes clear that exposure assessments can measure exposure for an individual or a population, typical and/or worst-case exposure, and can be directed at

[19] The Court considers supplemental and rebuttal reports in conjunction with the initial report for the purposes of determining reliability. *See Looksmart Grp., Inc. v. Microsoft Corp.*, No. 17-CV-04709-JST, 2019 WL 4009263, at *1 (N.D. Cal. Aug. 5, 2019) (considering a supplemental expert report for the purposes of determining the admissibility of the expert's opinions); *Baker v. SeaWorld Ent., Inc.*, 423 F. Supp. 3d 878, 910 (S.D. Cal. 2019) (considering both an expert initial and rebuttal report for the purposes of assessing reliability under 702); *Daniels v. Erie Insurance Group*, 291 F. Supp. 3d 835, 846 (M.D. Tenn. 2017) (considering both a revised and second revised report in a 702 analysis).

exposures that would occur based on a set of hypothetical conditions. *See* Ex. 36, Reference Manual at 519, 531, 537. In her professional work, Dr. Jones has done all of the above. *See* Def. Ex. 21, Jones Rpt. at 5; *see also* Ex. 49, Jones Rpt., Appendix A (Jones CV); Def. Ex. 41, Jones Vol. 1 Dep. at 115:14-116:6. It is well established that an expert can form opinions based on materials provided to them. *See United States v. Gomez*, 725 F.3d 1121, 1129 (9th Cir. 2013) ("As long as he is applying his training and experience to the sources before him and reaching an independent judgment, there will typically be no [] problem. The expert's opinion will be an original product that can be tested through cross-examination.") (internal citation omitted). Indeed, considering the unique specialties and expertise needed in complex toxic torts, it is better to have different experts focus on what they do best. Here, as in her professional work, Dr. Jones implemented the appropriate methodology for estimating average dietary exposure as would occur from the scenarios laid out in the hypotheticals—a methodology the Defendants have *not* challenged. What Dr. Jones did here is not meaningfully different from what Dr. Jones would be asked to do in a specific causation phase, except the actual products and consumption pattern at issue will not be hypothetical because they will be tied to a specific child's experience. Defendants cite no case law for the proposition that, in the field of exposure science, an expert cannot reliably calculate exposure based on inputs from an interested party. Indeed, in a non-bifurcated case, a plaintiff's exposure and consumption pattern will invariably come directly from the Plaintiff (or their parent), i.e., an interested party. *See, e.g.*, *In re Roundup*, 732 F. Supp. 3d at 1095 (finding exposure determined by plaintiff's discovery responses).

Similarly, whether the products on the menus are among the most or least contaminated products has absolutely no bearing on the reliability of the opinions offered by Dr. Jones, as she is not drawing any conclusions from her calculated exposure figures. Indeed, the consumption of different products and/or in different amounts and/or in different years will result in different lead and arsenic exposures that may be lower or higher than those calculated by Dr. Jones. But that is the stuff of specific, not general, causation. All that Dr. Jones is doing is providing a reliable estimate of lead and arsenic exposure from menus that were validated by a pediatric nutritionist (Ms. Barr), to then be interpreted by a panel of epidemiologists (Drs. Ritz, Gardener, and Hu) and toxicologists (Drs. Guilarte and Aschner). Dr. Jones does not claim that her exposure calculations are representative of

every person or Plaintiff's exposure to lead and arsenic from Defendants' products—such a claim would be impossible absent delving, headlong, into specific causation. To the contrary, Dr. Jones is clear that she is offering opinions about the exposures to lead and arsenic that would result from consumption of these specific menus of Defendants' baby food products. *See* Def. Ex. 21, Jones Rpt. at 76; *see also id*. at Tables 9, 15, 23, 29, 34, 41, and 47; Def. Ex. 23, Jones Rebuttal Rpt. at 6; Def. Ex. 41, Jones Vol. 1 Dep. at 153:12-154:24.

These exposures are all calculated based on real values provided in the analytical testing conducted by Defendants on their own products and ingredients that were consumed by real children during the specified time frames. Because there is no suggestion that Dr. Jones cherry-picked test results—for example, using only the highest test results to calculate mean values—and because Dr. Jones does not opine on whether the levels calculated are of a magnitude sufficient to cause neurodevelopmental harms, the cases cited by Defendants provide no support for Dr. Jones's exclusion. *See In re Bextra & Celebrex Mktg. Sales Practices & Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1176 (N.D. Cal. 2007) (finding that an expert who reaches his opinion by "cherry-picking observational studies that support his conclusion and rejecting or ignoring the great weight of the evidence that contradicts his conclusion" is not admissible); *Waymo LLC v. Uber Tech., Inc.*, 2017 WL 5148390, at *3-4, *8 (N.D. Cal. Nov. 6, 2017) (inactive lawyer and inactive CPA proffered as a damages expert excluded where he based unjust enrichment and reasonable royalty calculations on a single pitch slide and pieces of interrogatory responses that supported his opinion while ignoring the parts that contradicted it); *Rearden LLC v. Walt Disney Co.*, 2021 WL 6882227, at *7 (N.D. Cal. July 12, 2021) (excluding an expert for drawing sweeping conclusions about the alleged causal connection use of MOVA technology and a film's profitability based on a review of only 4 films that did not use MOVA technology and had low profitability). Here, as with the defendant's Rule 702 challenge in *Roundup*, the attack goes to the limits of the offered opinions, not the reliability of methodology, and any "limitations can be brought out on cross examination and made clear to the fact finder." *In re Roundup*, 732 F. Supp. 3d at 1096. In other words, it goes to weight, not admissibility.

Defendants' argument that Dr. Jones's calculation is a "black box into which data is fed at one end and from which an answer emerges at the other" is neither fair nor true. Def. Br. 2 at 16 (quoting

GPNE Corp. v. Apple, Inc., 2014 WL 1494247, at *4 (N.D. Cal. Apr. 16, 2014)). The method Dr. Jones uses is spelled out in detail in her reports. The exact data she used, including citations to the specific Bates numbered document used to collect that data, is provided. Her exact calculations are disclosed. This is anything but a black box. Defendants' citation to *GPNE Corp.* is completely inapposite. In *GPNE Corp. v. Apple, Inc.* the proffered expert testified that his estimate of a $1 per unit royalty from an $86 average net incremental profit was based on his professional judgment and 30 years of experience in the field. *See id*. at *4 (further reasoning that the court must be able to see the mechanisms of the methodology for the proffered opinions and cannot merely take an expert's word for a proposition). He did not provide any calculations, data, or even a principled formula that he used to estimate his $1 per unit royalty. *See id.* This "black box" stands in complete contrast to what Dr. Jones provides here. Her methodology and the mechanisms at play in her calculations of exposures and BLLs are painstakingly detailed in her report and numerous appendices and have been and can continue to be tested. *See* Def. Ex. 21, Jones Rpt.; *see also* Def. Ex. 23, Jones Rebuttal Rpt.; Def. Ex. 24, Jones Amended Rebuttal Rpt. That is more than sufficient under Rule 702.

b. Defendants' Attempt to Impugn Dr. Jones's Calculations by Comparing Her Results to National Averages Other Data Is Unavailing

Defendants next attack Dr. Jones because her estimated BLLs, based on lead exposure from Defendants' foods, are so high. *See* Def. Br. 2 at 19-20. Specifically, they argue that upon seeing the BLLs caused by Defendants' baby foods, Dr. Jones should have questioned the underlying menus. *See id*. at 19. But this is non-sequitur and misapprehends this lawsuit. The fact that Defendants' baby foods lead to very high BLLs is the entire point of the lawsuit. Infants who consumed Defendants' contaminated foods would be exposed to substantially greater levels of lead than "typical" children, which is why they went on to suffer brain injury and, ultimately, ASD and/or ADHD, while the neurotypical children did not. Seeing high BLLs based on the high intake of lead from Defendants' baby food should not cause Dr. Jones to second guess anything—it was *expected* considering how contaminated these baby foods were, based on Defendants' own testing. Moreover, how those calculated BLLs, based on those plausible exposure scenarios, are interpreted by others has nothing to do with the admissibility of her estimates using the IEUBK model. The question is

whether Dr. Jones's use of the EPA-created and validated IEUBK model "falls within the range of accepted standards governing how scientists conduct their research and reach their conclusions." *In re Roundup*, 390 F. Supp. 3d at 1130 (quotation marks omitted). And here, considering the IEUBK is a peer-reviewed and validated model used for specifically estimating an infant's BLLs, it is clear that Dr. Jones's analysis is entirely admissible.

Defendants cite a single study from 1978 that Defendants claim shows that, at the levels of lead intake calculated by Dr. Jones, the body excretes more lead than it absorbs. *See* Ex. 56, Ziegler, E, et al. (1978). This single fifty-year-old study, however, does not render Dr. Jones' estimate unreliable. Indeed, the IEUBK model, which is based on hundreds of peer-reviewed studies and validated work by the EPA over the past few decades, certainly controls for any issues related to absorption.[20] Dr. Jones's BLL calculations are based on a peer-reviewed and validated model that is broadly accepted as an appropriate method of calculating BLLs in young children. *See* Def. Ex. 21, Jones Rpt. at 10-11 (discussing the IEUBK model and references cited therein). One fifty-year-old study, untethered to any scientific explanation beyond attorney argument, does not defeat the predominance of the evidence supporting admissibility here.

Defendants' citations to the average and 97.5th percentile BLLs are also unavailing. Putting aside that the foods at issue here were severely contaminated with lead, which would cause high BLLs, Defendants' arguments miss the mark. The highest mean BLL estimated to result from lead exposure from Defendants' baby foods was from Hain, at 6.1 µg/dl. *See* Def. Ex. 21, Jones Rpt. at 50. Although that BLL is in the top few percent, there are many children whose BLLs exceed that value. *See* Ex. 57, Egan, et al. (2021). In other words, the data shows that Dr. Jones's BLL calculations are realistic, well below the highest levels *observed* in children. Moreover, as explained by Dr. Jones, in the field of exposure science, a high value is an indicator of right-skewed distributions of data—*i.e.*, a collection of self-identified plaintiffs that believe their exposure to

[20] *See* Ex. 104, EPA, *Frequent Questions from Risk Assessors on the Integrated Exposure Uptake Biokinetic (IEUBK) Model*, *General Questions* (Jan 2, 2025) ("The IEUBK model first quantifies the fraction of each media-specific intake that is bioaccessible or available for absorption in the gut. … The quantity of lead absorbed by the saturable pathway is a function of the total lead available in the gut, the fraction assumed to be absorbed via facilitated passive (PAF) diffusion, and the SATINTAKE variable, which is age specific.").

metals from baby food caused their injury—not faulty metrics. *See* Def. Ex. 42, Jones Vol. 2 Dep. at 566:15-567:16.

c. That Dr. Jones Revised Her Calculations Does Not Render Her Opinions Inadmissible

Under Rule 702, the Court must differentiate between "faulty methodology or theory as opposed to imperfect execution of laboratory techniques whose theoretical foundation is sufficiently accepted in the scientific community to pass muster under *Daubert*." *Hardeman*, 997 F.3d at 962 (quotation marks omitted). Here, where the Defendants' challenge—even if true—is based on data input errors, it provides no basis for exclusion.

Some of the calculations in Dr. Jones's original report were incorrect because there were data entry errors. Specifically, for certain Defendants' datasets, Dr. Jones inadvertently used duplicate testing results and a handful of results of ingredients / products that were not put to market. These errors were not deliberate. It was result of a massive dataset, the inscrutable nature of the testing documents produced, and several false interrogatory responses from Defendants. Those errors—which were all corrected—do not render her corrected calculations inadmissible. Dr. Jones's methodology is sound, a point laid bare by the fact that Defendants have not raised an issue with the way Dr. Jones calculated exposures to lead and arsenic. Defendants also do not contend that her methodology changed between reports. Instead, Defendants invite this Court to infer, from the fact that Dr. Jones revised her original dataset and subsequently updated her exposure calculations, that Dr. Jones's methodology suffers from unidentified "glaring flaws." Def. Br. 2 at 21. This sort of speculative challenge to Dr. Jones's analysis does not warrant exclusion.

Several of the "errors" in Dr. Jones's original calculations were due to the problematic data and discovery that Defendants provided. The Court instructed Defendants to provide to Plaintiffs, in an admissible format, the exact same information that Defendants had about their heavy metal test results so that Plaintiffs' experts could rely on that information and avoid "gotchas" during expert discovery. *See* Ex. 58, Jan. 23, 2025 CMC Tr. (Dkt. 362) at 15:6-16:18; *see also* PTO 12 (Dkt. 357) at 2. The Court was clear that while discovery is not perfect, it was the Court's expectation that the parties should have the same understanding as to which test results applied to each product and that Plaintiffs should know whatever Defendants knew about those test results. *See* Ex. 59, Feb. 27, 2025

CMC Tr. (Dkt. 432) at 116:18-25, 122:5-6.

Despite this order, Defendants presented an expert, Dr. Carolyn Scrafford (who is separately the subject of a motion to exclude), whose expert report was replete with various "gotchas" fed to her by Defendants. For example, Dr. Scrafford stated that Dr. Jones failed to exclude certain results from her analysis that pertained to products not sold, citing to documents which Defendants did *not* identify in response to interrogatory requests calling for the identification of same. *See* Ex. 48, Scrafford Rpt. at 46; *see also* Ex. 53, Beech-Nut Nutrition Cos First Supp. Resp. and Obj. Plas.' First Interrog. at 20-22, 31-32 (May 12, 2025). Dr. Scrafford was provided knowledge, after Dr. Jones had already completed her original analysis, which had not been provided to Plaintiffs. In another example, when counsel for Defendants confronted Dr. Jones with allegations that she failed to exclude certain results from her analysis that pertained to products not sold, Defendants did so by using documents produced after Dr. Jones's report was served (and shortly before Dr. Scrafford's report was served). *See* Def. Ex. 41, Jones Vol. 1 Dep. at 328:2-331:7; *see also* Ex. 60, Gerber's Production Ltr. (June 19, 2025). In yet another example, Plaintiffs' interrogatory requests asked Defendants to identify each *unique* heavy metal testing result for the products at issue and, after being ordered by this Court, they provided a list of Bates-numbered documents in response. *See e.g.*, Ex. 61, Hain's First Am. Resp. and Obj. Pls.' First Interrog. at 13-14, 16-17 (Mar. 26, 2025). Dr. Jones then relied on those sworn responses from Defendants when determining which testing results were, in fact, unique. *See* Ex. 41, Jones Rpt., Appendix D (Jones MCL) at 21; *see also* Def. Ex. 42, Jones Vol. 2 Dep. at 563:13-564:8. However, during Dr. Jones's deposition, Defense counsel confronted Dr. Jones with documents they claimed demonstrated that those previously identified as "unique" documents were, in fact, duplicates. *See* Ex. 61, Hain's First Am. Resp. and Obj. Pls.' First Interrog.at 13-14, 16-17; *see also* Def. Ex. 42, Jones Vol. 2 Dep. at 683:11-684:3. Each of these disclosures of information not provided in and/or controverted by their own sworn discovery responses. However, as soon as Dr. Jones learned that there were data entry issues with certain test results, she then corrected the data and re-ran the numbers—and they did not meaningfully impact the results (as would be expected considering the massive nature of the data at issue and the overwhelming majority of it being accurate). *Compare* Def. Ex. 21, Jones Rpt. at 35, 42, 50, 55, 61,

68, 75, *with* Def. Ex. 23, Jones Rebuttal Rpt. at 55, with Def. Ex. 24, Jones Amended Rebuttal Rpt. at 33. Indeed, considering the results did not meaningfully change despite the handful of errors identified by Defendants, it only confirms the lack of any systemic bias in the first place.

Moreover, it should be noted that the messy and inscrutable nature of their testing documents has forced Defendants to talk out of both sides of their mouth. For example, Dr. Jones attempted to remove duplicates from her analysis but did so imperfectly, removing only those duplicates flagged in the spreadsheets she received, believing (incorrectly) that the flagged entries were all of the duplicates. *See* Def. Ex. 21, Jones Rpt. at 12-13; *see also* Def. Ex. 41, Jones Vol. 1 Dep. at 43:20-45:11. However, Defense expert Dr. Robert Gibbons did the same thing. After receiving a spreadsheet of Defendants' heavy metal testing data from counsel with certain entries flagged as duplicates, Dr. Gibbons removed the flagged results, just as Dr. Jones did, but he did not further investigate whether there were additional duplicates before using that data to make his analysis. *See* Ex. 62, Gibbons Dep. at 101:22-102:15. And, as Dr. Jones's recalculation confirms, Dr. Gibbons acknowledged that inclusion of additional duplicates would not likely introduce bias or fundamentally change his results. *See id*. at 106:5-107:13; *see also* Ex. 63, Scrafford Vol. 1 Dep. at 53:3-11 (testifying that there are likely additional duplicates that her team did not flag). As another example, Defense expert Dr. Scrafford discusses Dr. Jones's inclusion of a <80 ppb lead result in her analysis as a data entry error that allegedly renders Dr. Jones's results unreliable. *See* Ex. 48, Scrafford Rpt. at 50; *see also* Ex. 63, Scrafford Vol. 1 Dep. at 90:23-91:16. Upon being confronted that she, too, had included this purported data entry error in the data she prepared (and on which Dr. Gibbons based his analysis), Dr. Scrafford suddenly could not say its inclusion rendered Dr. Gibbons's analysis unreliable. *See* Ex. 63, Scrafford Vol. 1 Dep. at 92:14-93:25; 94:1-19; *see also* Def. Ex. 25, Gibbons Rpt. at 22.

These are the kinds of "mistakes that may occur when large datasets are involved," as Defendants acknowledge. Def. Br. 2 at 21. They are not the kinds of methodological flaws that warrant exclusion under *Daubert*. Importantly, the data errors cited by Defendants have been *corrected*. Defendants point to nothing that suggests otherwise. Dr. Jones's most updated analysis—which is what Plaintiffs' experts rely on for the general causation analysis—is sufficiently reliable to

pass *Daubert*.

V. Plaintiffs' General Causation Experts Offer Admissible General Causation Opinions (Drs. Ritz, Hu, Gardener, Guilarte, Aschner, and Shapiro)

Plaintiffs submit Dr. Beate Ritz, Dr. Howard Hu, Dr. Hannah Gardener, Dr. Tomas Guilarte, Dr. Michael Aschner, and Dr. Kevin Shapiro.[21]

Dr. Beate Ritz is a Professor of Epidemiology at the UCLA Fielding School of Public Health, where she served as Vice Chair and Chair of the Epidemiology Department for a decade, and holds co-appointments in Environmental Health Sciences and Neurology at the UCLA School of Medicine. *See* Def. Ex. 1, Ritz Rpt. at 3; *see also* Ex. 64, Ritz Rpt., Appendix A (Ritz CV). She received an M.D. from the University of Hamburg/Germany, where she focused on psychiatry, as well as a doctoral degree from the University of Hamburg in Medical Sociology, and a doctoral degree in Epidemiology from UCLA. *See id.* For the past two decades, Dr. Ritz has been a principal investigator for numerous environmental epidemiology studies about the influence of pesticides and air pollutants—including toxic metals—on reproductive outcomes and autism in California. *See id.* Dr. Ritz has served on the editorial board of the journal *Epidemiology*, among others, and has served as the President of the International Society for Environmental Epidemiology (ISEE), which awarded her the Goldsmith Lifetime Career Award. *See id.* at 4. She has also received the Ken Rothman Career Achievement Award from the Society for Epidemiologic Research. *See id.*

Dr. Ritz concludes that early life exposure to lead and arsenic through Defendants' baby foods can cause a set of symptoms that can be diagnosed as ASD, and that the same lead exposure can cause a set of symptoms that can be diagnosed as ADHD. *See id*. at 5. Dr. Ritz also Dr. Ritz concludes that the beneficial nutrients in baby food products do not counteract the uptake or adverse neurological effects of metals to any clinically significant degree. *See id*. And, Dr. Ritz concludes that a child exposed to the levels of lead and arsenic through Defendants' baby foods identified in Dr.

[21] Plaintiffs also proffer the opinion of Dr. Dallas Reed, a board-certified geneticist and OB/GYN, who currently serves as Division Chief of Genetics and Director of Perinatal Genetics at Tufts Medical Center in Boston, Massachusetts. *See* Def. Ex. 17, Reed Rpt. at 1. Dr. Reed proffers expert opinion about the role of genetics in the etiology of ASD and, importantly, that environmental factors, including postnatal exposures, must be considered in assessing the potential causes of ASD. *See id*. at 1–2. Dr. Reed's opinions were not challenged and, thus, are not the subject of these *Daubert* motions.

Jones's report can "constitute substantial contributing factors in causing," and are sufficient to exacerbate, "brain injuries or neurodevelopmental harms that manifest as behaviors consistent with a diagnosis of ASD and/or ADHD." *See id.* at 70; *see also* Def. Ex. 2, Ritz Rebuttal Rpt. at 11.

Consistent with her other work and with the standards of her field, Dr. Ritz reached these conclusions by reviewing an extensive body of peer-reviewed scientific literature on the relationship between lead/arsenic exposure and ASD and between lead exposure and ADHD. *See e.g.*, Ex. 1, Ritz Rpt.; *see also* Ex. 65, Ritz Rpt., Appendix B (Ritz MCL). Her materials considered list includes 568 peer-reviewed articles, regulatory guidance, and Defendant product labels. *See id.* Dr. Ritz obtained these materials by conducting a literature search on PubMed and Google Scholar, databases that epidemiologists often use to assess causality. *See* Def. Ex. 1, Ritz Rpt. at 13. She further reviewed the published papers' citations and summary reports for any additional relevant articles. *See id.* at 14. In reviewing each article, she considered the quality of study design and analysis—including sample size, whether the study validly assessed exposure and outcome, and whether it avoided bias and confounding—to assess each study's validity and to determine how much weight to give to each study. *Id.* at 15. After considering each study and weighing its strengths and weaknesses, Dr. Ritz carefully applied the Bradford Hill factors to assess causality. *See id.* at 54-57, 61-63, 68-69.

Dr. Howard Hu is a Professor of Preventive Medicine at the Keck School of Medicine at the University of Southern California and an Adjunct Professor at the University of Michigan School of Public Health. *See* Def. Ex. 4, Hu Rpt. at 1; *see also* Ex. 66, Hu Rpt., Appendix A (Hu CV). He is a board-certified internist and preventive medicine specialist with a doctoral degree in epidemiology who has spent his career researching the potential impacts of exposure to lead on human health from the prenatal period through late adulthood. *See id*. After receiving his medical degree from the Albert Einstein College of Medicine, receiving his doctoral degree in epidemiology from the Harvard School of Public Health, and completing residencies at Boston City Hospital and the Harvard School of Public Health, Dr. Hu practiced medicine at Brigham and Women's Hospital, the University of Michigan Health System, Keck Medical Center, and elsewhere. *See id*. He was also the Founding Dean of the Dalla Lana School of Public Health at the University of Toronto and the Founding Director of the NIH/NIEHS Center for Children's Environmental Health at the Harvard School of

Public Health, and he has served as a member of the Board of Environmental Studies and Toxicology for the U.S. National Research Counsel, the Clean Air Scientific Advisory Committee Lead Review Panel for the EPA, and the Preventing Lead Exposure in Adults Workgroup for the CDC, among several other positions. *See id*. He was one of the three expert peer reviewers of the 2020 Toxicological Profile of Lead produced by the Agency for Toxic Substances and Disease Registry ("ATSDR") within the CDC. *See id*. Dr. Hu has published over 380 peer-reviewed articles, over half of which focus on lead exposure-related health outcomes. *See id*. at 2. The research he has led on environmental, nutritional, social, psychosocial, genetic and epigenetic determinants of impaired child development, including ASD and ADHD, has led to several awards, such as the Award of Excellence (for Research) from the American Public Health Association and the John Goldsmith Award from the International Society for Environmental Epidemiology. *See id*. at 1.

After a comprehensive review of the scientific literature, Dr. Hu concludes that lead is a cause or substantial contributing factor toward the development of ASD and ADHD; that lead exposure can also affect the severity of ASD and ADHD symptoms; and that the levels of lead in Defendants' baby food, as described by Dr. Jones, are capable of causing or exacerbating neurodevelopmental harms that can be diagnosed as ASD and ADHD. *See id.* at 8, 40; *accord.* Def. Ex. 5, Hu Rebuttal Rpt. at 1; Def. Ex. 6, Hu Am. Rebuttal Rpt. at 1. Dr. Hu reviewed 149 studies related to lead exposure and ASD and/or ADHD in children. *See* Ex. 67, Hu Rpt., Appendix D (Hu MCL). To find relevant literature, he conducted literature searches on PubMed and Scopus, as well as reports from international and national agencies related to the topic. *See* Def. Ex. 4, Hu Rpt. at 3. He evaluated the significance of each study by examining effect estimates and confidence intervals, potential bias, and the quality of methodology and study design, and he relied on the studies he determined to be the most rigorously conducted based on his over 30 years of experience researching lead toxicity. *See id.* Dr. Hu also provided tables of particular systematic reviews and meta-analyses relating to ADHD and ASD and discussed his rationale for weighing each study in his analysis. *See id.* at 14-17, 23-28. After compiling and considering all the materials in his materials considered list, Dr. Hu carefully applied the Bradford Hill factors to arrive at his causation opinions. *See id.* at 7, 19-20, 32-33.

Dr. Hannah Gardener is a Research Associate Professor at the Miller School of Medicine at

the University of Miami, where she has worked as an epidemiologist for over 18 years. *See* Def. Ex. 7, Gardener Rpt. at 3; *see also* Ex. 68, Gardener CV. She received her doctorate in Epidemiology, with a minor in Biostatistics, from the Harvard School of Public Health in 2007, where her doctoral work focused on prenatal, perinatal, and neonatal risk factors for autism. *See id*. Dr. Gardener has published over 139 peer-reviewed medical articles, with her research focusing on modifiable risk factors for a range of neurological outcomes. *See id.* She has also served as a consulting epidemiologist for several organizations, including the Clean Label Project. *See id.* Dr. Gardener has specifically been studying the burden of heavy metals in products since 2015. In 2019, before being retained as an expert, she published data on the concentrations of heavy metals in baby foods and infant formula. *See id.* at 4.

Dr. Gardener opines that lead accumulation in the body, including early life postnatal lead exposure through baby food, can interfere with early neurodevelopment and result in a set of behaviors that can be diagnosed as ASD and ADHD; that arsenic accumulation in the body, including early life postnatal arsenic exposure through baby food, can interfere with early neurodevelopment and result in a set of behaviors that can be diagnosed as ASD; and that the current data is insufficient to conclude that contemporaneous exposure to beneficial nutrients as found in baby foods (or supplementation with beneficial nutrients) offsets the uptake and/or adverse effects of neurotoxic metals in a clinically significant way. *See id.* at 5-7. Dr. Gardener also concludes that consumption of Defendants' commercial baby foods, at the levels estimated by Dr. Jones, can expose a child to heavy metals sufficient to cause ASD and/or ADHD. *See id.* at 88-89; *accord.* Def. Ex. 8, Gardener Rebuttal Rpt. at 1; Def. Ex. 9, Gardener Am. Rebuttal Rpt. at 1.

To arrive at her opinions, Dr. Gardener considered a vast body of epidemiological and toxicological data, conducted a systemic review of the literature, and applied the Bradford Hill factors. *See* Def. Ex. 7, Gardener Rpt. at 16-22. She conducted a search on PubMed for peer-reviewed studies on lead and arsenic in relation to ASD and lead in relation to ADHD, and she reviewed the studies' reference lists to identify any other potentially relevant studies. *See id.* at 16. Although she primarily focused on studies assessing heavy metal exposure from the postnatal period through childhood, for completeness she reviewed studies pertaining to prenatal heavy metal

exposures as well. *See id.* She also reviewed studies assessing the risk of neurotoxicity, including of ASD/ADHD, from metal exposure through food consumption; peer-reviewed literature addressing the potential impacts of nutrients, vitamins, and minerals in relation to heavy metal exposure; and animal models, *in vivo* studies, and *in vitro* studies examining the underlying biological mechanisms, as is customary for epidemiologists. *See id.* at 16–17. This process yielded over 550 documents on her materials considered list. *See* Ex. 69, Gardener MCL. In reviewing the literature, Dr. Gardener considered the timing of exposure, possible confounders, reverse causation, and the strengths and weaknesses of each study. *See* Def. Ex. 7, Gardener Rpt. at 17-22. She then carefully applied the Bradford Hill factors to arrive at her causation opinions. *See id.* at 76-78.

Dr. Tomas Guilarte has spent decades studying the neurotoxicological effects of heavy metals, including lead. *See* Def. Ex. 13, Guilarte Rpt. at 2; *see also* Ex. 70, Guilarte Rpt., Appendix A (Guilarte CV). He received his Ph.D. in Environmental Health from John Hopkins University and currently serves as the Dean of the Robert Stempel College of Public Health & Social Work at Florida International University, where he is a professor in the Department of Environmental Health Sciences and a visiting professor in Cognitive Neuroscience & Imaging in the Department of Physics. *See id*. at 2–3. In 1992, his lab discovered that lead inhibits NMDA receptors—critical for brain development and synapse formation—which led to the discovery of the link between lead's effect on brain development and increased risks of ASD and psychiatric diseases. *See id*. In 2023, he received a NIEHS grant to study the impact of chronic early life lead exposure on neurodevelopmental disorders. *See id*. Dr. Guilarte has been an editor of several respected peer-reviewed journals, including *NeuroToxicology*, *Toxicology and Applied Pharmacology*, and *Current Environmental Health Reports*, and has published over 165 peer-reviewed articles and 12 book chapters related to neurotoxicology and neuroscience. *See id.* at 6. He was recently recognized as one of the most impactful scholars in the specialty of lead poisoning by Scholar GPS. *See id.*

Dr. Guilarte concludes in his report that lead and arsenic can inflict permanent brain damage in young children, resulting in neurodevelopmental harms that can be diagnosed as ASD and/or ADHD; that even trace amounts of lead and arsenic consumed through baby food can cause disruption of critical enzymes and receptors in the brain, increasing the severity of the clinical

presentation of ASD and ADHD; that other nutrients found in baby food, such as calcium, iron, and zinc, do not counteract the neurotoxic effects of lead and arsenic on neurodevelopment; and that the levels of lead and arsenic that a child could consume through Defendants' baby foods, as identified by Dr. Jones, can substantially contribute to causing brain injuries or neurodevelopmental harms that can manifest as behaviors consistent with a diagnosis of ASD and/or ADHD, or can exacerbate ASD and ADHD symptoms in individuals diagnosed with these disorders. *See id.* at 53; *accord.* Def. Ex. 14, Guilarte Rebuttal Rpt. at 1; Def. Ex. 15, Guilarte Am. Rebuttal Rpt. at 1.

To reach these conclusions, Dr. Guilarte performed a systematic search of peer-reviewed literature related to ASD and ADHD on PubMed and Google Schoolar. *See* Def. Ex. 13, Guilarte Rpt. at 6. He reviewed, weighed, and relied upon over 300 peer-reviewed articles and regulatory publications. *See* Ex. 71, Guilarte Rpt., Appendix B (Guilarte MCL). Similar to the methodologies that he uses in his own research, and as is typical in the field of toxicology, Dr. Guilarte performed a weight of the evidence approach, assessing animal data, mechanistic data, and epidemiological data to arrive at his conclusions. *See* Def. Ex. 13, Guilarte Rpt. at 7.

Dr. Michael Aschner is the Harold and Muriel Block Endowed Chair at the Albert Einstein College of Medicine, where he also serves as a professor in multiple departments, including Molecular Pharmacology, Neuroscience, and Pediatrics, and as Investigator for the Rose F. Kennedy Intellectual and Developmental Disabilities Research Center. *See* Def. Ex. 10, Aschner Rpt. at 1; *see also* Ex. 72, Aschner CV. The focus of his forty-plus year career has been the interaction between genetics and the environment in triggering brain diseases at all life-stages, with a particular specialty in heavy metals. *See id.* at 2. He has published approximately 1,125 peer-reviewed articles and 120 book chapters and has co-edited several books and authoritative texts on toxicology. *Id.* at 4. His work has been cited over 82,000 times. *See id*. He has received the Merit Award from the Society of Toxicology (SOT), the highest recognition in toxicology, as well as the Career Achievement Award from the Metal Specialty Section and the Distinguished Neurotoxicologist Award from the Neurotoxicology Specialty Section of SOT. *See id*. at 2. He has also served on and chaired numerous national and international committees, including with the National Institutes of Health, EPA, ATSDR, the Department of Defense, Health Canada, and the European Food Safety Authority.

See id. at 3. Years before becoming involved in this litigation, Dr. Aschner published numerous studies identifying heavy metals as causes of autism, specifically noting in one peer-reviewed paper: "Pb [lead] exposure has been named as one of the causes of ASD…this review discusses reports highlighting neurotoxic metals (specifically, lead, [and] mercury…as environmental risk factors in the etiology of ASD." Ex. 73, Ijomone, O., et al. (2020) at 1; *see also* Ex. 74, Aluko, O., et al. (2021) at 4 ("Metals may disrupt the strictly regulated brain development process, resulting in neurodevelopment disorders, such as ASD."); Ex. 34, Goel, A., et al. (2021) at 1 ("[T]he current data and trends suggest a potential strong role for lead in ASD."). Dr. Aschner, thus, has already published and subjected his opinions related to lead causing ASD to peer-review.

In his report here, Dr. Aschner concludes that exposure to heavy metals can interfere with neurodevelopment and thus cause ASD/ADHD, that such exposures through baby foods can interfere with neurodevelopment and cause ASD/ADHD, and that the available scientific data does not support the conclusion that the nutritional composition of baby food (or food in general) affects the uptake or neurotoxicity of heavy metals to a clinically significant degree. *See* Def. Ex. 10, Aschner Rpt. at 6-8. He also opines that a child's exposure to lead and arsenic from Defendants' products, as illustrated in Dr. Jones' dose calculations, would constitute a meaningful dose of exposure and could constitute a substantial factor in causing a child's ASD and/or ADHD diagnoses. *See id.* at 72; *accord* Def. Ex. 11, Aschner Rebuttal Rpt. at 1; Def. Ex. 12, Aschner Rebuttal Rpt. at 1.

To reach his opinions, Dr. Aschner analyzed the toxicological and epidemiological evidence, drew from his scientific experience and expertise, and used a weight of evidence approach that toxicologists often employ. *See* Def. Ex. 10, Aschner Rpt. at 10. This approach "involves the rigorous assessment of all toxicological, mechanistic, and epidemiological data to form a judgment regarding the likelihood that the exposure, i.e., lead and/or arsenic, can cause an outcome, i.e., ASD and/or ADHD." *See id*. Dr. Aschner gathered relevant literature by conducting a search on PubMed for articles relating to keywords for lead, arsenic, neurodevelopment, ASD, ADHD, and neurotoxicity, as well as literature about the relationship between nutrients, minerals, vitamins and neurotoxic heavy metals and literature about the presence and effect of heavy metals in commercial baby food. *See id.* His materials considered list includes 513 articles that he reviewed, analyzed, and

considered, and his resulting conclusions are supported by a wealth of epidemiological data and the toxicological profile of these heavy metals. *See e.g.,* Def. Ex. 10, Aschner Rpt.; *see also*, Ex. 75, Aschner MCL.

Dr. Kevin Shapiro is a board-certified neurologist with a specialty in pediatric neurology and decades of experience diagnosing and treating children with ASD, ADHD, and a host of other neurodevelopmental conditions. *See* Def. Ex. 16, Shapiro Rpt. at 1; *see also* Ex. 76, Shapiro CV. He received his MD and a Ph.D. in Psychology (cognition, brain, and behavior) from Harvard Medical School and Harvard University, respectively, and completed his internship and residency in pediatric medicine at Boston Children's Hospital, followed by a residency in neurology and child neurology at Massachusetts General Hospital. *See id*. at 1–2. He now serves as the Medical Director and Clinical Executive for Research at Cortica Healthcare, is a member of the Neurology staff at Valley Children's Hospital in Madera, California, and maintains privileges as an affiliate staff member at Children's Hospital Los Angeles and Rady Children's Hospital in San Diego. *See id.* He also regularly treats pediatric and young adult patients in Lifetime Neurodevelopmental Care at Community Regional Medical Center in Fresno, California, and Kaiser Permanente Oakland Medical Center in Oakland, California. *See id.* As a result of his extensive training, clinical practice, and research, Dr. Shapiro has an intimate familiarity with the etiology of ASD/ADHD and with the impact of heavy metal exposure, particularly lead and arsenic, on neurocognitive development in children.

In his report, Dr. Shapiro offers several opinions relating to biological mechanisms. *See id*. at 4-5. He opines that the pathogenesis of ASD and ADHD is attributable to genetic factors, environmental risk factors, and gene-environment interactions. *See id*. He opines that exposure to heavy metals has been associated with biological processes that manifest in ASD and ADHD, including reductions in intelligence, behavioral problems, and other symptoms of ADHD. *See id*. He opines that the biological pathways implicated in the pathogenesis of ASD and ADHD overlap with the pathways by which lead and arsenic affect neuronal function and development both *in vivo* and *in vitro*. *See id*. And he opines that current scientific evidence does not support the notion that the nutritional composition of food mitigates the uptake or neurotoxic effects of lead and arsenic. *Id*.

To arrive at his opinions, Dr. Shapiro employed a methodology typical of what he would apply in his clinical practice. *See id*. at 5-6. He conducted an independent review of the scientific literature by searching online databases, including PubMed and Google Scholar, for the available literature regarding those effects of metal exposure on the developing brain that are relevant to the relationship between metal exposure and neurodevelopmental conditions such as ASD/ADHD and the significance of such exposures through baby food. *See id.; see also* Ex. 77, Shapiro MCL. After weighing the strengths and weaknesses of the data—and relying on his significant clinical experience with diagnosing and treating children with ASD/ADHD and his research experience in the etiology and pathogenesis of ASD and ADHD—Dr. Shapiro reached his conclusions based on the aggregate scientific evidence that he reviewed in light of his clinical expertise, experience, and judgment. *Id*.

A. Each Experts' General Causation Opinion Meets the Elements of Rule 702

The elements of Rule 702 are easily satisfied for each expert.

First, as demonstrated in each report, each expert possesses "scientific, technical, or other specialized knowledge" that will "help the trier of fact to understand the evidence" and resolve general causation. FED. R. EVID. 702(a). Plaintiffs' experts are eminently qualified to proffer opinions that will aid the jury's charge.

Second, these reports and testimony demonstrate that each expert's "testimony is based on sufficient facts or data[.]" FED. R. EVID. 702(b). Each of Plaintiffs' general causation experts began their analysis by conducting a comprehensive search of relevant, reliable scientific literature published in peer-reviewed journals. Consistent with their academic and professional practices, each searched PubMed, the world's leading database for peer-reviewed science, to identify the scope of potentially relevant literature; some also used additional databases, such as Google Scholar and Scopus, to conduct additional searches. After identifying the scope of potentially relevant material, each of Plaintiffs' experts reviewed the material to identify those publications whose relevance and reliability merited in-depth evaluation. Each expert considered all available epidemiology, animal studies, and mechanistic studies before arriving at their respective opinions. *See* Def. Ex. 1, Ritz Rpt. at 13-17; *see also* Def. Ex. 4, Hu Rpt. at 3-8; Def. Ex. 7, Gardener Rpt. at 16-26; Def. Ex. 13, Guilarte Rpt. at 6-7; Def. Ex. 10, Aschner Rpt. at 10-13; and Def. Ex. 16, Shapiro Rpt. at 5-7. Thus,

it is clear, these experts' opinions are supported by sufficient facts and data.

Third, these reports and testimony demonstrate that each expert's opinions are "the product of reliable principles and methods." FED. R. EVID. 702(c). Each expert applied well-accepted methodologies to reach their opinions. First, they all conducted comprehensive literature reviews, reviewing and considering hundreds of studies (noted above). Then, after reviewing the literature, each expert utilized a specific method to assess causation (or, for Dr. Shapiro, biological plausibility). Plaintiffs' epidemiologists (Drs. Ritz, Hu, and Gardener) applied the Bradford-Hill framework, *see* Def. Ex. 1, Ritz Rpt. at 5-6; *see also* Def. Ex. 4, Hu Rpt. at 7; Def. Ex. 7, Gardener Rpt. at 5-6; Plaintiffs' toxicologists (Drs. Guilarte and Aschner) applied the weight of the evidence methodology, *see* Def. Ex. 13, Guilarte Rpt. at 8; *see also* Def. Ex. 10, Aschner Rpt. at 10-13; and Plaintiffs' clinician (Dr. Shapiro) conducted a comprehensive literature review, *see* Def. Ex. 16, Shapiro Rpt. at 5-6.

"The Bradford Hill criteria are nine factors generally accepted as relevant to assessing causation." *Hardeman*, 997 F.3d at 953 n. 2. Epidemiologists apply the Bradford Hill framework to determine whether an association observed in epidemiological research between an exposure and a health outcome is causal. *See* Ex. 36, Reference Manual at 549, 600. As Dr. Hill explained:

> None of my nine viewpoints can bring indisputable evidence for or against the cause and- effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question - is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Ex. 78, Hill (1965) at 299. The Bradford Hill framework is "well accepted in the medical field for making causal judgments." *Wendell v. GlaxoSmithKline LLC*, 858 F.3d 1227, 1235 n. 4 (9th Cir. 2017); *see also* Ex. 36, Reference Manual at 597-600. Thus, "[t]o the extent the *Daubert* question is whether consideration of the Bradford Hill factors is a reliable method for determining causation as a general matter, the answer is yes." *In re Roundup*, 390 F. Supp. 3d at 1130 (citations omitted).

Consistent with general practice among toxicologists, both of Plaintiffs' toxicologist witnesses – Drs. Guilarte and Aschner – apply the weight of the evidence methodology. *See* Def. Ex. 13, Guilarte Rpt. at 8; *see also* Def. Ex. 10, Aschner Rpt. at 10-13. Both the EPA and the European Food Safety Authority ("EFSA") utilize the weight of the evidence methodology to evaluate causal

relationships between exposures and health outcomes. *General Elec. Co. v. Joiner*, 522 U.S. 136, 152-54 (1997) (Stevens, J., concurring) (noting EPA's use of the weight of the evidence methodology); *see also* Ex. 79, Hardy, A., et al. (2017) at 5-6 (discussing use of weight of the evidence methodology in EFSA analyses). Unsurprisingly, then, "[n]o serious argument can be made that the weight of the evidence approach is inherently unreliable." *Milward v. Acuity Specialty Prods. Grp., Inc.*, 639 F.3d 11, 18 (1st Cir. 2011); *see also, e.g., Waite v. All Acquisition Corp.*, 194 F. Supp. 3d 1298, 1313 (S.D. Fla. 2016). Indeed, in *Joiner*, Justice Stevens approvingly noted the Court of Appeals' decision that "a 'weight of the evidence' methodology [is] scientifically acceptable" to evaluate general causation. 522 U.S. at 153-54 (Stevens, J., concurring). Justice Stevens found that "[i]t is not intrinsically 'unscientific' for experienced professionals to arrive at a conclusion by weighing all available scientific evidence—this is not the sort of 'junk science' with which *Daubert* was concerned." *Id.* at 153; *see also In re Abilify (Aripiprazole) Prod. Liab. Litig.*, 299 F. Supp. 3d 1291, 1312 (N.D. Fl. 2018) ("Where an expert is found to have applied the 'weight of the evidence' approach with 'the same level of intellectual rigor' used by experts in the field, his general causation opinion typically will be deemed reliable and admissible.").

Dr. Shapiro, a clinician and researcher, applied a methodology accepted among his professional peers and courts in the Ninth Circuit. Dr. Shapiro is not proffering a general causation opinion, *per se*. Instead, he proffers opinions about the biological plausibility of lead and arsenic interfering with neurodevelopment in ASD children. A clinician's application of their "education, training and experience, knowledge of the pertinent medical literature and [their] knowledge of the epidemiology, diagnosis, and natural history of" the health outcome of interest is an admissible approach to evaluating biological plausibility in the context of causal relationships between exposures and health outcomes. *Wendell*, 858 F.3d at 1234; *see Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1228-30 (9th Cir. 1998) (reversing exclusion of clinician researcher who reached general causation opinion by conducting literature review in light of clinical education and experience). Dr. Shapiro reviewed the epidemiological literature for evidence of relevant associations and considered the biological plausibility of causal explanations for those associations in light of his expertise in diagnosing, treating, and researching childhood neurological issues, including a particular focus on

autism. Each of Plaintiffs' general causation experts began their analysis by conducting a comprehensive search of relevant, reliable scientific literature published in peer-reviewed journals. Consistent with their academic and professional practices, each searched PubMed, the world's leading database for peer-reviewed science, to identify the scope of potentially-relevant literature; some also used additional databases, such as Google Scholar and Scopus, to conduct additional searches. After identifying the scope of potentially-relevant material, each of Plaintiffs' experts reviewed the material to identify those publications whose relevance and reliability merited in-depth evaluation. *See* Def. Ex. 1, Ritz Rpt. at 13-17; *see also* Def. Ex. 4, Hu Rpt. at 3-8; Def. Ex. 7, Gardener Rpt. at 16-21; Def Ex. 13, Guilarte Rpt. at 6-7; Def. Ex. 10, Aschner Rpt. at 10-13; Def. Ex. 16, Shapiro Rpt. at 5-7. Each then evaluated the strengths and weaknesses, import, and evidentiary value of each publication pursuant to their field of expertise's causation methodology. Courts regularly admit expert testimony that relies on literature review. *See, e.g., In re Roundup*, 390 F. Supp. 3d at 1130-31, 1139 (admitting experts who begin their general causation analysis by "conduct[ing] a literature search to identify the relevant epidemiology evidence."); *In re Zimmer Nexgen Knee Implant Prods. Liab. Litig.*, 2015 WL 5050214, at *3 (N.D. Ill. Aug. 25, 2015) (admissible literature review "uses formal search methods to allow a researcher to obtain a neutral 'snapshot' of the existing research on a particular question."). It is well-established that "epidemiological, animal, and cell" studies, such as those on which Plaintiffs' experts rely, are reliable sources of information when considering causation questions. *Hardeman*, 997 F.3d at 963.

On this record, the evidence supports the conclusion that these experts' opinions are the "product of reliable principles and methods." FED. R. EVID. 702(c).

And finally, a preponderance of the evidence establishes that each expert's general causation "opinion reflects a reliable application of the principles and methods to the facts of the case FED. R. EVID. 702(d). Each expert considered each of these varying types of scientific evidence in light of their chosen methodology, and none of them ignored publications that reached ultimate conclusions contrary to their own. *See, e.g.,* Def. Ex. 1, Ritz Rpt. at 47-48 (noting that Guo et al. (2019) meta-analysis did not find statistically significant differences in hair lead levels in ASD cases and controls); *see also* Def. Ex. 7, Gardener Rpt. at 42 (noting Wang et al. (2019)'s characterization of

evidence of lead-ASD association as "inconsistent"). Indeed, as noted above, there are numerous peer-reviewed studies that specifically reach causation opinions about the ability of lead and arsenic to cause ASD and/or ADHD. *See supra* Part III.D-F. That Plaintiffs' experts reach conclusions that independent scientists have in peer-reviewed studies have also reached, speaks loudly for the proposition that Plaintiffs' experts' opinions—putting aside if they are ultimately correct—fall within the "bounds of what can be concluded from a reliable application of the expert's basis and methodology." Fed. R. Evid. 702 (Advisory Committee note to 2023 amendment). In fact, the FDA already concluded that the lead ingested in baby food is capable of harming "children's health and development, specifically the brain and nervous system." Ex. 15, FDA Lead Guidance at 4. They caution that lead exposures from baby food above the IRL "serves as a useful benchmark in evaluating the potential for adverse effects of dietary lead exposure, such as the potential for neurodevelopmental effects." *Id.* at 6.

Plaintiffs' experts Drs. Ritz, Hu, Gardener, Guilarte and Aschner, each in keeping with their respective educational and professional backgrounds, reviewed and interpreted Dr. Jones's exposure calculations as well. Drs. Ritz, Hu, and Gardener considered Dr. Jones's calculations in conjunction with their Bradford Hill analyses. *See, e.g.,* Def. Ex. 1, Ritz Rpt. at 70; *see also* Def. Ex. 7, Gardener Rpt. at 7; Def. Ex. 4, Hu Rpt. at 40; Def. Ex. 2, Ritz Rebuttal Rpt., at 11; Def. Ex. 8, Gardener Rebuttal Rpt. at 1; Def. Ex. 5, Hu Rebuttal Rpt. at 1. Drs. Guilarte and Aschner considered Dr. Jones's calculations in their weight of the evidence methodologies. *See* Def. Ex. 13, Guilarte Rpt. at 53; *see also* Def. Ex. 10, Aschner Rpt. at 71-72; Def. Ex. 14, Guilarte Rebuttal Rpt. at 1; Def. Ex. 11, Aschner Rebuttal Rpt. at 1. These estimates provide assurance that the lead and arsenic exposures that a child could be exposed to from eating menus of each Defendants' baby foods are capable of causing ASD and/or ADHD.

Each of Plaintiffs' experts also reliably opines that vitamins and minerals found in baby food products do not prevent or offset the neurotoxicity of the lead and arsenic present in those products. As noted above, where the relevant literature does not support an association between an exposure and an outcome, the Bradford Hill framework is not used to evaluate a potential causal relationship. *See, e.g.,* Ex. 36, Reference Manual at 597 ("*Once an association has been found* . . ., researchers

consider whether the association reflects a true cause-effect relationship") (emphasis added); *In re Roundup*, 390 F. Supp. 3d at 1130 ("a reliable assessment that an association between [an exposure] and [an endpoint] exists in the epidemiological literature is a prerequisite to application of the [Bradford Hill] criteria") (citing Ex. 36, Reference Manual at 597). As Plaintiffs' experts explain, they considered the scientific literature relevant to the question of whether vitamins and minerals present in baby food products prevent or offset neurotoxic harm caused by heavy metals. *See, e.g.,* Def. Ex. 10, Aschner Rpt. at 73-92; *see also* Def. Ex. 16, Shapiro Rpt. at 32-33. They each conclude, based on analyses they explain in their reports and depositions, that this literature does not support an association between consumption of these vitamins and minerals and protection against the neurotoxicity of heavy metals, with the possible exception of nutrient-deficient children. *Id*.

All told, on this record, a preponderance of the evidence makes it clear that Plaintiffs' experts' general causation opinions reflect "a reliable application of the principles and methods to the facts of the case." FED. R. EVID. 702(d).

B. Defendants' Challenges to Plaintiffs' General Causation Experts Are Unavailing

Defendants raise a series of challenges to Plaintiffs' general causation experts. They are, generally, divided into three sections. First, Defendants argue that Plaintiffs' experts cannot establish general causation because, according to them, "no science" supports any association between baby food and ASD or ADHD. They make this argument by asserting, despite Plaintiffs' allegations to the contrary, that the "toxic agent" at issue in this case is baby food, not lead or arsenic. Indeed, only by ignoring science related to lead and arsenic—including the mountain of studies associating lead and arsenic to ASD and ADHD and the robust testing evidence proving that Defendants baby foods contain high levels of these neurotoxins—can Defendants claim there is "no science." But, as explained below, these arguments are misleading and, factually, wrong. Second, Defendants argue that Plaintiffs' experts fail to link the estimated lead and arsenic doses in their baby foods to any increased risk of ASD or ADHD. But described below, that is simply untrue—Plaintiffs' experts carefully and explicitly link the levels of lead and arsenic found in Defendants' baby foods to an increased risk of ASD and/or ADHD. Third, Defendants take aim at the underlying science itself, arguing that the peer-reviewed studies directly linking lead and arsenic to ASD and/or ADHD,

including those peer-review studies that explicitly reach causation conclusions, are unreliable. These arguments, as discussed below, fall apart upon inspection.

1. There Is Substantial Science Associating the Lead and Arsenic Found in Defendants' Baby Foods to an Increased Risk of Developing ASD and/or ADHD

a. There Are Numerous Studies Examining Whether the "Toxic Agent"—here Lead and Arsenic—Cause ASD and/or ADHD

Defendants argue that general causation fails, across the board, because there are no studies looking at whether baby food consumption increases the risk of ASD or ADHD. *See* Def. Br. 3 at 8-9. This is a complete red herring as there are hundreds of studies examining whether the toxic agents at issue, *i.e.*, lead and arsenic, are associated with ASD and/or ADHD. And, as explained by Plaintiffs' experts, there is consensus within the scientific community that heavy metal exposure harms early neurodevelopment *irrespective of the source*. *See* Ex. 22, Shapiro Vol. 1 L.R. Dep. at 94:9-16, 284:19-286:8 ("[T]he route of exposure is not relevant … So whether you eat them in food or … are exposed to lead paint or soil or environmental pollution, that the method by which you're exposed to those metals doesn't seem to have a significant impact on the adverse outcomes…"); *accord* Def. Ex. 46, Gardener L.R. Dep. at 118:20-119:18; *see also* Ex. 26, ATSDR Lead Profile at 12 ("[t]he primary systemic toxic effects of Pb are the same regardless of the route of entry into the body.") Def. Ex. 1, Ritz Rpt. at 25; Def. Ex. 4, Hu Rpt. at 36; Def. Ex. 7, Gardener Rpt. at 89-90; Def. Ex. 10, Aschner Rpt. at 15-16; Def. Ex. 35, Guilarte Dep. at 367:22-368:18. In fact, dietary heavy metal exposure often poses a higher health risk than other sources because "for the majority of children age 1-6, food is the primary contributor to blood lead levels." Def. Ex. 7, Gardener Rpt. at 85 (citing Ex. 80, Zartarian, V., et al. (2017)). Indeed, Plaintiffs' experts rely upon a veritable mountain of evidence which shows that exposure to heavy metals in early life, including through baby food consumption, can interfere with neurodevelopment sufficient to cause ASD/ADHD. *See generally* Def. Ex. 1, Ritz Rpt.; Def. Ex. 4, Hu Rpt.; Def. Ex. 7, Gardener Rpt.; Def. Ex. 10, Aschner Rpt.; Def. Ex. 13, Guilarte Rpt.; Def. Ex. 16, Shapiro Rpt. And that view is also reached by independent scientists. For example, in a recent publication titled, "Investigating the role of food pollutants in autism spectrum disorder: a comprehensive analysis of heavy metals, pesticides, and mycotoxins" they concluded:

> This review sought to explore the ***influence of food contaminants on ASD*** by thoroughly examining the effects of heavy metals ... Through a comprehensive analysis of existing research and scientific data, several key findings have emerged. Firstly, it is apparent that ***heavy metals like Pb … and As have been implicated in the onset and severity of ASD.*** The harmful impacts of these metals on the developing brain are well-documented, and ***their presence in food sources raises concerns regarding their potential contribution to the prevalence of ASD***.

Ex. 81, Nehzomi & Shirani (2024) at 19 (emphasis added).

b. The General Benefits of Food Are Irrelevant to Whether Food Contaminated with Lead and Arsenic Can Cause Brain Damage

Defendants assert that Plaintiffs' experts' methodologies are unreliable just because the fruits and vegetables in baby food are "healthy for brain development[.]" Def. Br. 3 at 9-10. This is akin to arguing that water contaminated with poison is safe because water "is good for humans." The potential for certain nutrients to *mitigate* the neurotoxic effects of lead and arsenic is relevant—and that is precisely what Plaintiffs' experts considered. But simply saying "nutritious food"[22] is "good for babies" ignores the real question: whether otherwise nutritious food *contaminated with toxic heavy metals* increases the risk of neurodevelopmental harm. *See* Def. Ex. 43, Ritz L.R. Dep at 49:7-13 ("Of course, they are beneficial, ***but whether they are beneficial in comparison to the neurotoxic agent***.") (emphasis added); *accord* Def. Ex. 35, Guilarte Dep. at 322:23-323:8.

It is telling that Defendants invoke vague assertions by regulatory bodies instead of the underlying science. Def. Br. 3 at 10. The studies undercut their argument. As one study observed:

> ***Despite recommendations*** to consume iron-, calcium-,and vitamin C-rich foods for managing blood lead levels (BLLs), ***limited evidence*** exists on how specific foods affect children's BLLs… Among young children, consumption of iron-, calcium-, and vitamin C-rich foods showed ***weak or no association with BLLs***…Our study, along with the previous studies, indicates that although nutrient-based recommendations may be useful when a single toxicant is under consideration, they may ***not be optimal*** when translated into food-based guidelines within a context of ***multiple toxicants***.

Ex. 82, Desai, et al. (2021) at 471, 477 (emphasis added). And, in another study:

> There are no safe BLLs and levels <5 µg/dL are associated with a range of adverse neurobehavioral effects, including lower IQ and behavioral problems…Children are

[22] Plenty of Defendants' most profitable products, like infant rice cereal, are not particularly nutritious. Indeed, the American Academy of Pediatrics recommends that parents avoid rice cereal altogether. *See* Ex. 98, Trisha Korioth, *Parent Plus: Limit infants' exposure to arsenic by feeding a variety of grains*, American Academy of Pediatrics (May 19, 2016).

> vulnerable to dietary lead exposure because they could absorb >40 % of the lead they ingest…The mostly null associations observed in this study between diet quality and BLLs are contrary to our hypotheses; they ***do not suggest that higher quality diets may protect children by lowering BLLs***.

Ex. 83, Kordas, et al. (2024) at 2, 7 (emphasis added).

Defendants attack Plaintiffs' experts because they treat lead and arsenic exposure from food the same as exposure from other sources. *See* Def. Br. 3 at 10–15. But, as Dr. Hu and others explain,

> [O]nce lead is absorbed from baby food, it would not be expected to behave or carry risks of causing adverse neurodevelopmental outcomes, including ASD, any differently from lead that is absorbed from any other source. In other words, I'm not aware of any research indicating that lead absorbed from different dietary sources or from different sources of intake (i.e., inhalation v. ingestion) would be expected to behave differently depending on the source.

Def. Ex. 4, Hu Rpt. at 35; *see also* Ex. 22, Shapiro Vol. 1 L.R. Dep. at 94:9-16, 284:19-286:8 ("[W]hat I'm aware of are studies that establish a relationship between exposure to arsenic and lead…and autism. And I'm also aware of studies that establish that the ***route of exposure is not relevant***…the method by which you're exposed to those metals ***doesn't seem to have a significant impact on the adverse outcomes***…") (emphasis added). This point is echoed by the ATSDR. *See* Ex. 26, ATSDR Lead Profile at 12 ("The primary systemic toxic effects of Pb are the same regardless of the route of entry into the body."); *see also* Def. Ex. 47, Aschner L.R. Dep. at 165:19-166:5 ("[T]he source of the food or the source of the exposure makes absolutely no difference. What determines the toxicity of lead … arsenic is…the levels of these metals in the blood and in other tissues, and in this case the brain."). When a child is exposed to metals through consumption of baby foods, the potential neurodevelopmental harm of such exposure is no different than metal exposure from any other source, whether it comes from paint chips, water, rice cereal, or a baby food pouch.

Indeed, the literature assessing ASD/ADHD risk from heavy metal exposure involved children who, by definition, consumed "whole" food containing beneficial nutrients as well as neurotoxic heavy metals (as well as children from different parts of the world consuming a variety of diets). *See* Def. Ex. 43, Ritz L.R. Dep. at 46:24-47:6 ("[The studies] are measuring heavy metals in the blood, urine, hair or nails, and that's usually from ***multiple sources, including food***. And food can be a very considerable source, because that's what babies have to eat in order to grow.") (emphasis added). That the literature observes a consistent association between lead and arsenic

exposure (including through food) and an increased risk of neurodevelopmental conditions—among which include ASD and ADHD—underscores Plaintiffs' experts' conclusions that the nutritional composition of food does not generally clinically affect the adverse effects of toxic heavy metals.

c. Plaintiffs' Experts Reliably Reviewed the Literature to Determine Whether the Beneficial Nutrients Found in Defendants' Baby Foods Would Mitigate the Adverse Effects of Lead and Arsenic

Defendants repeatedly refer to "whole food." Where a "whole food" contains neurotoxins like lead and arsenic, analyzing its risks and benefits does not merely involve analyzing whether unspecified nutrients, vitamins, and minerals are "nutritious." The analysis must consider whether the "nutritious" components can mitigate, prevent, or offset the known neurotoxic effect of the lead and arsenic the whole food contains. This is exactly what Plaintiffs' experts did—specifically evaluating whether the nutritional composition of baby food (or food in general) can impact the uptake or adverse neurological effects of heavy metals. *See* Def. Ex. 1, Ritz Rpt. at 5, 24-37; *see also* Def. Ex. 7, Gardener Rpt. at 6-7, 80-109; Def. Ex. 10, Aschner Rpt. at 7-8, 65-93; Def. Ex. 4, Hu Rpt. at 34-35; Def. Ex. 13, Guilarte Rpt. at 7, 32-35; Def. Ex. 16, Shapiro Rpt. at 31-36. The relevant literature evaluates the extent to which specific nutrients affect metal uptake and neurotoxicity—for example, Vitamin C mitigates lead and not, as Defendants presumably would have it, whether oranges mitigate lead.) There is nothing else in food beyond the specific nutrients, vitamins, and minerals (considered by Plaintiffs' experts) hypothesized to affect the uptake or adverse effects of heavy metals. And, as explained in Plaintiffs' experts' reports, these studies do not support the proposition that the nutritional components of food have a clinical impact on the uptake or neurological effects of heavy metals. The CDC agrees. *See* Ex. 99, CDC (2002) at 64, 65, 67, 68 (finding a lack of evidence that nutritional components of food affect heavy metal uptake or toxicity).

The exception to this, as Plaintiffs' experts acknowledge, is that certain beneficial nutrients can impact metal absorption and intake in children who are nutritionally deficient. *See e.g.*, Def. Ex. 26, Ritz Dep. at 242:4-8 ("From the studies that I saw, it worked in children who were deficient in iron or other nutrients, but it did not necessarily work in children who were not deficient."), 88:21-90:7, 67:8-25; *accord* Def. Ex. 4, Hu Rpt. at 34-35; Def. Ex. 7, Gardener Rpt. at 109 ("[W]hile adequate consumption of nutrients has been recommended for children exposed to heavy metals,

nothing in the literature indicates that exposure to heavy metals through food containing nutrients results in neutralization, or even reduction, of the neurotoxic effect of these metals in a clinically meaningful way."); *accord* Def. Ex. 10, Aschner Rpt. at 93; Def. Ex. 13, Guilarte Rpt. at 34; Def. Ex. 16, Shapiro Rpt. at 32-33. In other words, certain minerals and vitamins can affect lead and arsenic absorption when a child is nutritionally deficient, but there does not appear to be any clinically meaningful effect of these beneficial nutrients on lead ingestion in an otherwise nutritionally sufficient child.

Defendants nonetheless argue that "none of Plaintiffs' experts' opinions reliably account for the ways that exposure to lead and arsenic in healthy foods containing essential nutrients differs from exposure to the same heavy metals through exposure sources like water, air, or soil." Def. Br. 3 at 13. But, this is simply false.

First, Defendants claim that Dr. Ritz did not consider the extent to which "eating fruits and vegetables with trace amounts of heavy metals at the same levels found in baby food would result in a net reduction in oxidative stress in the body or a net increase in oxidative stress in the body." *Id*. But instead of quoting Dr. Ritz, Defendants cite their *own lawyer's question* during Dr. Ritz's deposition. *Id*. (citing Def. Ex. 26, Ritz Dep. at 238:13-239:4). Contrary to Defendants' assertion, Dr. Ritz testified that the vitamin content of Defendants' baby food products is not sufficiently high to mitigate any oxidative stress caused by metal exposure through the foods. *See* Def. Ex. 26, Ritz Dep. at 227:17-228:1 ("Well, what I saw is the content of baby food in terms of vitamins was not very impressive…And so I don't think that there is a sufficient amount…that, you know, would actually be balancing out the oxidative stress."); *see also id*. at 236:18-22.

Second, Defendants cite Dr. Gardener's state court testimony for the proposition that Dr. Gardener did not consider the antioxidant effects of nutrients in baby food. *See* Def. Br. 3 at 13. To be clear, however, Dr. Gardener's response was in the context of discussing a study which found that "toxic metal exposure can decrease antioxidant effects and -- as well as alter DNA methylation as potential pathways through which the toxic metal exposure increases the risk of autism." Def. Ex. 46, Gardener L.R. Dep. at 162:16-20. The critical point Dr. Gardener made is that while cellular-level competition between nutrients and heavy metals exists, this does not translate to clinically

significant protection that would negate the harmful effects of heavy metal exposure on a population level. *See id*. at 139:3-6 ("Because on a cellular level, there is the competition, but that doesn't mean that…in a clinical, whole-body level that there's actually a meaningful association.").

Defendants also argue that Dr. Gardener did not consider food as part of her general causation opinions. *See* Def. Br. 3 at 14-15. This is absurd. No fewer than *30 pages* of Dr. Gardener's report are devoted to evaluating the issue of whether the nutritional composition of baby food (or food in general) affects the uptake or adverse neurological effects of metals. *See* Def. Ex. 7, Gardener Rpt. at 80-109. And, at deposition, Dr. Gardener explained that the available studies focus on the constituent nutrients found in "whole food." *See* Def. Ex. 46, Gardener L.R. Dep. at 135:21-136:23, 138:17-139:6. As she explained (but Defendants omit), the issue is not whether nutrients are "neuroprotective" but whether they mitigate the uptake or adverse effects of neurotoxic heavy metals that can be present in food: "[M]y report has to do with heavy metals. And so the impacts of…these foods *independent of heavy metals* is beyond the scope of…my report." *Id*. at 145:19-22 (emphasis added).

Third, Defendants accuse Dr. Hu of not "investigat[ing] the composition of Defendants' foods, review any of the relevant literature, or do anything else to determine whether the net impact of baby food on neurodevelopment is positive or negative, including with respect to autism/ADHD diagnosis risk." Def. Br. 3 at 13-14. Not true. The question is not whether eating food, in general, correlates with ASD/ADHD risk (such a study would not be possible because all humans eat food), but whether the nutritional composition of Defendants' baby foods impacts metal uptake/adverse effects. Dr. Hu explained that he considered "the levels of contaminants that are found in a number of different baby products…as well as contents of the nutrients that are contained in those baby products." Def. Ex. 29, Hu Dep. at 20:10-16; *accord id*. at 20:20-21:3 And, after considering the complex baby food mixture (the nutrients and neurotoxic metals) and the available scientific data on the interactions between the nutritional composition of the food and metals, Dr. Hu concluded there is "no evidence that diets that have recommended levels of iron and calcium, or higher-than recommended levels of iron and calcium, or higher-than-recommended levels of any other nutrient, will cause decreases in lead absorption among children that result in lead absorption rates that are

substantially different than the 40-50% absorption rate of ingested lead." Def. Ex. 4, Hu Rpt. at 34.

Defendants also assert that Dr. Hu's approach was inconsistent with one of his publications where he studied interactions between the nutritional composition of food and heavy metals. *See* Def. Br. 3 at 14. Not so. When shown his prior study (Wang, et al. (2017)), Dr. Hu explained that although there is a difference in effect when multiple nutrients are considered, he "would not consider these as the kinds of interactions or combined effects that would…undercut…the effect of lead, particularly once it's absorbed from the gastrointestinal tract into blood."). Def. Ex. 29, Hu Dep. at 188:11-21.

Fourth, Defendants claim that Drs. Aschner, Guilarte and Shapiro did not "assess the potential interactive effect of healthy nutrients and heavy metals when consumed in food." Def. Br. 3 at 15. Again, this is simply not true. Dr. Aschner focused almost *40 pages* of his report on the issue of metal exposure through baby food and the interaction between the nutritional composition of the foods and heavy metals. *See* Def. Ex. 10, Aschner Rpt. at 6, 59-93. Defendants cite *none* of this analysis. And, at deposition, Dr. Aschner explained that he "focused mainly on the divalent anions given the nature of these metals…Lead is primarily divalent, so there's a good reason to look at iron. There's a good reason to look at calcium." Def. Ex. 47, Aschner L.R. Dep. at 263:9-23; *see also* 264:190-25. He was not confronted with any studies or body of data that he failed to consider. Rather, Defendants quote one sentence (out of Dr. Aschner's multiple-volume depositions) wherein Dr. Aschner merely testified that he did not consider, in isolation from metal exposure, the extent to which consuming food is good for the brain. *See* Def. Br. 3 at 15. This does not mean that Dr. Aschner ignored any components of the food. Defendants omit Dr. Aschner's testimony that the nutritional components of baby food "that are discussed in my review are those that I could find that directly relate to the absorption of different metals and whether they diminish or augment the effects of these metals, arsenic and lead. So if there was no literature on a given nutrient and arsenic absorption or the effects of arsenic, it is obviously not in my report…" Def. Ex. 34, Aschner Dep. at 85:3-22.

Similarly, Dr. Guilarte testified that he has "a whole section in my expert report related to the whole perception that essential metals can compete with lead for absorption in the intestines." Def.

Ex. 35, Guilarte Dep. at 171:19-172:6; *see also id*. at 330:7-15, 327:21-328:4. And, with respect to Defendants' counsel convoluted questions regarding "whole food," Dr. Guilarte testified that "I don't think that study has been done," but explained that, based on the available mechanistic evidence, the nutritional components of food are insufficient to mitigate the adverse effects of lead exposure. *Id*. at 364:6-19, 365:1-3; *see also id.* at 369:12-20 ("My opinion about mechanism again is related to lead and arsenic…If it's in regular food and you're getting it in sufficient amounts, the mechanism should be applied.").

Lastly, Dr. Shapiro's report is clear that he thoroughly considered the interaction between the nutritional composition of baby food and heavy metals. *See* Def. Ex. 16, Shapiro Rpt. at 32-34. Defendants claim that Dr. Shapiro is not aware which specific products are at issue in this litigation. *See* Def. Br. 3 at 15. That is beside the point. To arrive at his conclusions, Dr. Shapiro "searched for and summarized ***all the literature I could find*** that was relevant to the question at hand." Def. Ex. 36, Shapiro Vol. 1 Dep. at 177:4-6. Notably, the levels of the nutritional components of the foods in the studies "are much higher than what we are typically exposed to in the diet," *id*. at 226:7-9, underscoring Plaintiffs' experts' opinions that, based on the available data, there is no reason to conclude that the nutritional composition of the baby foods at issue—which contain far less beneficial nutrients—would have an effect on the adverse effects of metal exposure through consumption of the foods. *See* Ex. 22, Shapiro Vol. 1 L.R. Dep. at 365:14-366:1 (noting that "the data in general are not sufficient to make a conclusion that…there is a significant impact on metal-induced neurotoxicity with increased nutrient consumption in non-deficient individuals.").

d. Plaintiffs' Experts Did Not "Ignore" Any Relevant Data on Food

Defendants next attack Plaintiffs' experts by claiming they "ignore the extensive body of literature involving food." Def. Br. 3 at 16. This is simply false.

First, "body of literature involving food" is a meaningless concept. This is demonstrated by Defendants' invocation of only *two* studies: Rahbar (2021) and Rahbar (2022). *See id*. at 16. Defendants claim that Plaintiffs' experts did not consider these studies in rendering their opinions. Not true—they are discussed or listed in each report. *See* Def. Ex. 10, Aschner Rpt. at 30; *see also* Def. Ex. 13, Guilarte Rpt. at 30; Ex. 69, Gardener MCL at 34; Ex. 65, Ritz MCL at 32; Def. Ex. 1,

Ritz Rpt. at 52; Def. Ex. 26, Ritz Dep. at 105:2-111:3; Ex. 77, Shapiro MCL at 45. But, even if Plaintiffs' experts had not considered these studies, that would not be fatal to their opinions because Rahbar (2021) and Rahbar (2022) are not particularly informative of the issues in this litigation. Contrary to Defendants' assertion, Rahbar (2022) did not evaluate interactions between neurotoxic metals and beneficial nutrients vis-à-vis neurological outcomes. As Dr. Ritz explained: "It actually has ***nothing to do with it***. It has to do with ASD cases having peculiar diets that might exclude certain types of food. Whether these kids had higher exposure prior to being diagnosed, we don't know about here." Def. Ex. 43, Ritz L.R. Dep. at 158:11-20 (emphasis added); *see also id.* at 149:11-151:9, 154:7-18, 154:20-155:22, 156:15-157:2. And, with respect to Rahbar (2021), Dr. Ritz explained: "**Q**. And in these Rahbar studies, typically developing children had higher levels of heavy metals than children with autism. Is that right? **A**. Well, they're eating the kind of vegetables that contain lead, so I wouldn't be surprised if they had higher levels. But in these adjusted geometric means, ***there is really no difference***." Def. Ex. 26, Ritz Dep. at 110:18-111:2 (emphasis added); *see also id.* at 109:19-110:3. To the extent that Defendants even have a challenge to Dr. Ritz' opinions based on their misunderstood reading of Rahbar, it goes to weight, not admissibility.

Dr. Aschner likewise provided a cogent explanation of his consideration of the Rahbar data, which Defendants do not quote. *See* Def. Ex. 34, Aschner Dep. at 288:15-289:10 ("There's no analysis of the levels of lead or arsenic in any of these foods…"). Rahbar (2021) and Rahbar (2022) are simply inapposite.

Second, Defendants claim that "Plaintiffs' experts also ignore studies showing that children who are introduced earlier to solid foods, including commercial baby foods, do *not* have an increased risk of subsequent autism diagnosis compared to children who receive their calories solely from breast milk and formula for a longer period of time." Def. Br. 3 at 17. Again, this is not true. In support, Defendants cite three inapposite studies: Emond (2010), Campbell (2024), and Xiang (2023).[23] *Id*. To be clear, *none* of these three studies are relevant—indeed, not even the Defendants' experts discuss these studies. With respect to Xiang (2023), Dr. Ritz explained that the results are

[23] Defendants only examined Dr. Ritz on two of these studies (Xiang and Emond) and did not examine any of Plaintiffs' other experts regarding these studies.

likely due to reverse causation and that it does not stand for the proposition that Defendants assert:

> **Q**. Would you agree that these results suggest that there may be a link between the delayed introduction of foods and autism? **A**. Well, what I suggested was that ***we don't know from this data alone*** because children's behavior and their food acceptance may already be a symptom for those 10 percent or 20 percent who did not accept solid foods. It may already be a symptom of their ASD. ***It doesn't say that if you would force-feed them complimentary foods you could prevent ASD***….**Q**. Do they mention anything about reverse causation? **A**. ***Yes, actually they do***…[quotes study]:'This may be because the severity of the neurodevelopmental delay affects the timing of the introduction of complimentary foods in infants.'

Def. Ex. 26, Ritz Dep. at 133:19-135:20 (emphasis added). With respect to Emond (2010), Dr. Ritz explained that Defendants misinterpret the study as concluding that diet was related to subjects' ASD diagnoses: "These 79 children with autism have a different dietary pattern from children who don't have autism. And that's nothing new because we know that ASD children are picky eaters. It doesn't say whether the diet caused or not caused or prevented their ASD." Def. Ex. 26, Ritz Dep. at 277:5-12.[24] This is not, as Defendants claim, "advocacy" (Def. Br. 3 at 17) but a scientist critically engaging with the data and providing clear scientific explanations as why the studies do not represent what Defendants lawyers (not their experts) think about them. There is also no "inconsistency" in Dr. Ritz's opinions. Defendants argue that "Dr. Ritz is willing to testify generally that children who eat Defendants' baby foods starting four to six months after birth can have autism because they ate baby food.[25] But when shown data suggesting the opposite, her conflicting response is that the autism must have developed before the baby food eating began." Def. Br. 3 at 17. This misses the mark. Dr. Ritz explained that neither Xiang nor Emond discuss levels of dietary heavy metal exposure, or how such exposure levels interact with the nutritional components of the food to impact the children's neurodevelopment. These studies suggest that a higher percentage of ASD children begin eating baby foods (as opposed to bottles) a few months later than neurotypical children. So what? Unless there is some data indicating what levels of lead and arsenic each group was being

[24] Campbell (2024) is merely about "feeding difficulties" experienced by children with autism. It does not address the impact of the nutritional composition of food or metals on neurodevelopment.
[25] This is, obviously, an intentionally oversimplistic representation of Dr. Ritz's opinion. No one claims that eating baby food, uncontaminated with lead or arsenic, causes ASD. However, consuming lead and arsenic in baby food, during early life, does increase the risk of developing ASD and ADHD, as confirmed by hundreds of studies demonstrating, empirically, that early life exposure to these metals leads to brain damage and, later in life, ASD and/or ADHD diagnoses.

exposed to, these studies do not provide much insight into the causation question. It says nothing about food preferences or, more importantly, food consumption patterns of children before developing ASD. And, Dr. Ritz expressly considered these studies and explains exactly why they do not move the needle. Such an analysis does not make her methodology in any way unreliable.

e. Plaintiffs' Experts Considered the Relevant Nutrient Literature

Defendants next criticize Plaintiffs' experts' reliance on studies that assessed the interaction between single nutrients and heavy metals. *See* Def. Br. 3 at 18-20. But, Plaintiffs' experts did not base their opinions solely on "single nutrient" or "supplementation" studies, but considered "whole food" studies where available, even though the majority of the available literature is in the form of single-nutrient or supplementation studies. *See* Def. Ex. 46, Gardener L.R. Dep. at 136:2-23, 111:18-24, 138:17-139:6; *see also* Def. Ex. 43, Ritz L.R. Dep. at 103:18-25; Def. Ex. 7, at 100-01 (discussing Desai et al. (2021)); *accord* Def. Ex. 10, at 86-87. In fact, the small number of available "whole food" studies support the opinions of Plaintiffs' experts. *See* Ex. 82, Desai, et al. (2021) at 471, 477 (quoted *supra*); *see also* Ex. 83, Kordas, et al. (2024) at 2, 7 ("The mostly null associations observed in this study between diet quality and BLLs are contrary to our hypotheses; ***they do not suggest that higher quality diets may protect children by lowering BLLs***.") (emphasis added); Ex. 84, Ma, et al. 2025 at 9 (consumers of brown rice were at increased risk of arsenic toxicity notwithstanding the presence of beneficial nutrients in such diets). And, as Dr. Aschner explained:

> [M]ultiple studies conducted in different parts of the world under diverse exposure scenarios demonstrate that exposure to heavy metals in early life can interfere with neurodevelopment … This causal trend under various exposure scenarios is illustrative of the fact that heavy metal exposure, regardless of source, can … interfere with the developing brain and result in a set of behaviors that can be diagnosed as ASD and ADHD.

Def. Ex. 10, Aschner Rpt. at 67. Tellingly, Defendants do not point to a single "whole diet and food study" that Plaintiffs' experts "failed to consider" which shows that the nutritional composition of food affects the uptake or adverse effects of heavy metals to a clinically significant degree. Defendants' other arguments are equally unavailing.

First, Defendants argue that "these studies do not consider food as a whole, but instead attempt to assess the impact of singular nutrients or vitamins such as iron, Vitamin D, or calcium, when food has dozens of nutrients and vitamins and other constituent parts." Def. Br. 3 at 19. Of

course, Defendants do not identify any of the "dozens" of other "nutrients and vitamins" that they contend are in food and somehow affect metal uptake that Plaintiffs' experts supposedly failed to consider. Instead, Defendants gloss over this point. For example, Defendants cite Dr. Hu's testimony that it is important to consider "whole food" but then omit the critical part of his deposition where he explicitly testified that he considered "whole food" before reaching his conclusions. *See* Def. Ex. 29, Hu Dep. at 188:11-16 ("I would not consider these as the kinds of interactions or combined effects that would…undercut…the effect of lead, particularly once it's absorbed from the gastrointestinal tract into blood."). Defendants also accuse Dr. Ritz of not having considered arsenic absorption, excretion, and storage, Def. Br. 3 at 20, when, to the contrary, several pages of her report contain discussions of studies addressing this very issue. *See* Def. Ex. 1, Ritz Rpt. at 31-38; *see also* Def. Ex. 43, Ritz L.R. Dep. at 103:18-25.

Second, Defendants argue that "the single nutrient studies that Plaintiffs' experts rely on evaluate reduction in blood and urine lead levels, but do not assess the impact of these nutrients on neurodevelopmental endpoints, much less autism and ADHD." Def. Br. 3 at 20. This, again, misses the point. Plaintiffs' experts are here to provide opinions about whether the lead and arsenic in Defendants' baby foods can cause neurodevelopmental harm, not whether certain nutrients in food promote better neurodevelopmental outcomes. And, determining whether the "beneficial" nutrients offset lead and arsenic effects is exactly what these single nutrient studies do—they specifically isolate and determine whether these nutrients have any impact on the toxic effects of lead or arsenic.

Third, Defendants contend that "the designs of these studies, which investigate nutrient effects in children with high levels of environmental exposure to lead or arsenic, cannot speak to what happens at a dose and ratio of nutrients and metals found in food." Def. Br. 3 at 20. Defendants, again, miss the mark. As noted, the levels of the nutritional components of the foods in such studies "are much higher than what we are typically exposed to in the diet," Ex. 22, Shapiro Vol. 1 L.R. Dep. at 226:7-9, so there is no reason to conclude that the nutritional composition of the baby foods at issue would have a meaningful effect on the adverse effects of metal exposure through consumption of the foods. *Id.* at 365:14-366:1; *accord* Def. Ex. 34, Aschner Dep. at 303:17-20. And, as Dr. Ritz explained, the results of the studies are inconsistent due to widely disparate study designs

and populations, most of the positive results are observed in children with nutritional deficiencies, and the doses of nutrients observed to not have a meaningful effect on metal absorption were significantly higher than the levels of such nutrients found in baby food products. *See* Def. Ex. 43, Ritz L.R. Dep. at 67:8-20, 72:7-17, 75:23-76:10, 77:2-15, 80:5-15, 91:21-92:4, 105:16-24, 111:20-112:7, 116:19-117:3, 117:4-10. But, that aside, the whole food studies relied upon by Plaintiffs' experts demonstrate that the nutritional composition of food does not meaningfully counteract the uptake or adverse effects of metals in children who are exposed to both the nutrients and metals through "whole food[.]" *See* Def. Ex. 34, Aschner Dep. at 302:24-303:5 ("[A]s I said before…the *Kordas studies looked at different diets and blood levels in kids, and they found out that, irrespective of the nature of the diets, the lead levels are the same*. So I don't think that supplementing with calcium or iron is going to make any difference.") (emphasis added); *see also* Def. Ex. 8, Gardener Rebuttal Rpt. MCL at 19, 9 (identifying Kordas (2024) study and Desai (2021) study that evaluated metal and nutrient exposure through whole food); Def. Ex. 7, Gardener Rpt. at 100-101 (discussing Desai); Def. Ex. 2, Ritz Rebuttal Rpt. MCL at 22 (referencing Kordas (2024)); *accord* Def. Ex. 14, Guilarte Rebuttal Rpt. MCL at 10; Def. Ex. 5, Hu Rebuttal Rpt. MCL at 8. The entire premise of Defendants challenge—that Plaintiffs' experts did not consider whole food studies and, as such, cannot reliably opine that the beneficial nutrients in baby food do not offset the adverse impacts of lead and arsenic is simply untrue. The literature, which Plaintiffs' experts considered, indicates that there is simply no valid evidence that these nutrients, whether found in whole foods or in isolation, stop lead and arsenic from causing brain damage.

2. Plaintiffs' Experts Do Not Proffer an "Any Dose, Any Exposure, Any Window" Opinion

Defendants claim that Plaintiffs' experts did not consider dose in reaching their causation opinion. *See* Def. Br. 3 at 21-25. This is demonstrably false.

a. Plaintiffs' Experts Proffer Admissible Opinions Regarding the Causal Significance of Exposure to the Doses of Heavy Metals from Consumption of Defendants' Baby Foods

Defendants argue that Plaintiffs' experts do not connect the doses of exposure from consumption of Defendants' baby foods (calculated by Dr. Jones) to an increased risk of developing ASD/ADHD. *See* Def. Br. 3 at 21-25. This is simply not correct. Indeed, Defendants' argument is

bereft of citation to Plaintiffs' experts' reports and complete deposition testimonies and is based upon a distorted understanding of Plaintiffs' experts' opinions regarding the causal significance of dose.

As an initial matter, Plaintiffs' general causation experts provided extensive and reasoned discussions of: 1) the principles of threshold doses, and the widely recognized maxim of "no safe level of exposure" to heavy metals; and 2) the causal significance, on a population level, of heavy metal exposure from consumption of Defendants' baby food products.

First, as Plaintiffs' experts explain, the absence of a "threshold dose" for exposure to heavy metals and ASD/ADHD does not preclude a finding that exposure to specific doses of heavy metals from baby food consumption increases the risk of ASD/ADHD and does not mean that "any" exposure causes injury. *See* Def. Ex. 7, Gardener Rpt. at 31 (explaining the difference between dose response and threshold dose); *see also* Def. Ex. 1, Ritz Rpt. at 37-39 ("[A] threshold dose is inapplicable due to the stochastic nature of the outcome at issue…a threshold dose is the concept that there exists a defined dose of exposure to a toxin below which there is no risk of a specific outcome…***on a population level***. However, the probability that a particular exposure will result in harm to an individual does not turn on whether that individual was exposed to a pre-defined threshold dose of the toxin.") (emphasis added); Def. Ex. 10, Aschner Rpt. at 8-10 ("[I]t is well established that cigarette smoke causes lung cancer. However, no threshold dose has ever been established for cigarette smoke, whereby smoking poses zero risk. This lack of identifiable threshold dose has not prevented toxicologists or epidemiologists to reach a causation conclusion."); *see also* Def. Ex. 4, Hu Rpt. at 36-37; Def. Ex. 13, Guilarte Rpt. at 8, 41-42.[26] In other words, the dose makes the poison ***depending on the individual***.

Second, although a threshold dose has not been identified for the association between metal exposure and ASD/ADHD, the scientific evidence (with which Defendants do not engage) discussed by Plaintiffs' experts evinces augmented risk for developing ASD/ADHD following exposure to specific doses of heavy metals. And exposure to the doses of metal from consumption of

[26] Defendants' own experts agree. For example, when Defendants' expert Elise Robinson was asked, "you don't have to have a threshold dose or a threshold dose model to be able to reach a causal conclusion between an exposure and an outcome; right?" she did not mince words: "No, that ***would be a silly requirement*** because most dose-response relationships don't follow a threshold model…" Ex. 86, Robinson Dep. at 123:8–20 (emphasis added).

Defendants' baby foods is above the doses observed in the epidemiological data as increasing the risk of ASD/ADHD. Specifically, two studies (discussed in Plaintiffs' experts' reports) indicate that blood lead levels ranging from 1 µg/dL (*i.e.*, above the mean for U.S. children of 0.670 µg/dL) are associated with an increased risk of ASD/ADHD. *See, e.g.*, Def. Ex. 85, Kim, et al. (2016) (observing that "[e]ven low blood lead concentrations" are associated with autistic behaviors – with one-unit incremental increases in blood lead measures associated with poorer scores on ASD symptom questionnaires); Ex. 85, Ha, et al. (2008) (blood lead levels of 1-<1.5, -<2.5, -<3.5, and >3.5 ug/dl associated with increased risk of ADHD). These studies were explicitly considered by Plaintiffs' experts. *See* Def. Ex. 10, Aschner Rpt. at 34-35, 55-56; *see also* Def. Ex. 7, Gardener Rpt. at 22-23, 47, 53, 55; Def. Ex. 13, Guilarte Rpt. at 20; Def. Ex. 4, Hu Rpt. at 28-29; Def. Ex. 1, Ritz Rpt. at 20, 38, 46, 54; Ex. 75, Aschner MCL at 14; Ex. 69, Gardener MCL at 20; Def. Ex. 14, Guilarte Rebuttal Rpt. MCL at 7; Def. Ex. 5, Hu Rebuttal MCL at 6; Ex. 65, Ritz MCL at 17. And, as shown in Dr. Jones's BLL estimates, each Defendant's baby foods were capable of causing an infant's BLL to reach at least 1 µg/dL from consumption of those baby foods alone. *See* Def. Ex. 21, Jones Rpt. at 35, 42, 50, 55, 61, 68, 75; *see also* Def. Ex. 23, Jones Rebuttal Rpt. at 55; Def. Ex. 24, Jones Amended Rebuttal Rpt. at 33. Clearly, these lead levels are capable of exposing infants to levels of lead that the epidemiology shows is associated with ASD and ADHD.

The FDA also recognizes that exposures to lead above the agency's 2.2 ug/day indicates "when lead exposure from food contributes significantly to BLLs." Ex. 9, Flannery & Middleton (2022) at 2; *see also* Ex. 15, FDA Lead Guidance (2025); Def. Ex. 10, Aschner Rpt. at 17-18; Def. Ex. 7, Gardener Rpt. at 35-36, 88-89, 111-114; Def. Ex. 14, Guilarte Rebuttal Rpt. MCL at 6; Def. Ex. 5, Hu Rebuttal Rpt. MCL at 5; Def. Ex. 1, Ritz Rpt. at 24-25. According to the FDA, when an infant consumes more than the IRL from baby food, *i.e.*, 2.2 ug/day of lead, it serves as a "benchmark in evaluating the potential for adverse effects of dietary lead exposure, such as the potential for neurodevelopmental effects." Ex. 15, FDA Lead Guidance (2025) at 6. And here, as shown in Dr. Jones's calculations, each Defendants' baby foods were capable of exposing an infant to more than 2.2 ug/day of lead—again, this would lead exposure on top of any other lead exposure the child may have had from other sources. *See* Def. Ex. 21, Jones Rpt. at 35, 42, 50, 55, 61, 68, 75; *see also* Def.

Ex. 23, Jones Rebuttal Rpt. at 55; Def. Ex. 24, Jones Amended Rebuttal Rpt. at 33.

Regarding arsenic, one study conducted by the same company that employs Defendants' expert, Dr. Scrafford, estimated that arsenic exposures above 0.4-1 µg/kg/day have been reliably associated with neurotoxicity in pediatric populations. *See* Ex. 87, Tsuji, et al. (2015); *see also* Ex. 88, Zhu, et al. (2024) (Neurobehavioral effects of arsenic detected at concentrations as low as 0.81 µg/L); Def. Ex. 10, Aschner Rpt. at 69-70; Def. Ex. 7, Gardener Rpt. at 84; Ex. 71, Guilarte Rpt., Appendix B (Guilarte MCL) at 15; Ex. 65, Ritz MCL at 39. So, if a child weighs 10 kg, any exposure in excess of 4– 10 µg/day would increase risk. And, as Dr. Jones's estimates show, every menu of Defendants' baby foods could exceed that level of exposure. *See* Def. Ex. 21, Jones Rpt. at 35, 42, 50, 55, 61, 68, 75; *see also* Def. Ex. 23, Jones Rebuttal Rpt. at 55; Def. Ex. 24, Jones Amended Rebuttal Rpt. at 33.

Based on review of these doses as compared to the doses reported in the literature, Plaintiffs' experts were easily able to conclude that "that [the levels of exposure from Defendants' products] meet or exceed levels associated with ASD and/or ADHD in the epidemiological literature I analyzed in forming the conclusions described in this report." Def. Ex. 1, Ritz Rpt. at 6, 70; *see also* Def. Ex. 4, Hu Rpt. at 40; Def. Ex. 7, Gardener Rpt. at 90; Def. Ex. 10, Aschner Rpt. at 75; Def. Ex. 13, Guilarte Rpt. at 53. Contrary to Defendants' assertion, this is far from "say so" or "any dose goes." Rather, Plaintiffs' experts opine on the causal significance, on a population level, of exposure to the doses of metals from consumption of Defendants' baby foods based on the available scientific data.

Third, Defendants assert that when "pressed in deposition to elaborate on how they were relying on Dr. Jones's estimates, Plaintiffs' experts demurred and said that those estimates were not necessary to their ultimate causation opinions because there is 'no safe level' of lead or arsenic." Def. Br. 3 at 22. This is false. Defendants' only support for this assertion is a reference to the deposition of Dr. Shapiro, which Defendants do not even quote. As an initial matter, Defendants attack an opinion (the causal significance of the dose of exposure through consumption of Defendants' baby foods as calculated by Dr. Jones) that Dr. Shapiro (unlike Drs. Ritz, Gardener, Aschner, Hu, and Guilarte) is ***not*** giving. In fact, Dr. Shapiro confirms as much in the portion of his deposition cited by Defendants: "**Q**. And I assume just for specificity with respect to Dr. Jones, am I

correct that you did not rely on Dr. Jones' analysis, including any exposure amounts that were included in that report? **A**. I had not seen that report at the time I prepared my report, and my – in this context, my opinions are not tied to specific exposure amounts." Def. Ex. 36, Shapiro Vol. 1 Dep. at 14:13-21. In other words, in the portion of Dr. Shapiro's testimony cited by Defendants, he merely reiterated that "the scientific consensus in the scientific literature" holds that there is no safe level of exposure to heavy metals. *Id*. at 63:16-23. This colloquy is inapposite to Defendants' assertion that "the experts seem to be retreating to claim that any dose of lead or arsenic is capable of causing autism or ADHD, irrespective of the levels estimated by Dr. Jones." Def. Br. 3 at 22. Dr. Shapiro has no opinions about Dr. Jones's numbers.

Defendants then, again, misquote Dr. Shapiro to argue that Plaintiffs' experts opine that "any" exposure causes ASD/ADHD. *Id*. To be clear, Defendants asked Dr. Shapiro whether consuming "any food with any amount of arsenic is capable of causing autism," including "one carrot that has 1 microgram of arsenic." Def. Ex. 36, Shapiro Vol. 1 Dep. at 146:24-147:3, 148:11-18. Dr. Shapiro reasonably explained that while no threshold dose has been identified, on a population level, below which arsenic does not cause harm, "the degree to which it contributes to the outcome in any ***particular child*** depends on a number of factors, including that ***child's susceptibility*** to toxicity, other factors that might have contributed to the same outcome and stochastic events, such as how that particular molecule of arsenic interacts with DNA or other cellular processes." *Id*. at 147:6-148:9 (emphasis added). And he explained that he could not "rule out" 1 microgram causing harm based on what we know about arsenic, but that it is an absurd hypothetical to entertain without more information regarding the exposed child. *Id*. at 148:11-152:4 ("[I]n a ***hypothetical universe*** in which we're conversing, that you could ***imagine*** some circumstance in which that could occur.") (emphasis added). Defendants' argument is therefore meritless. Plaintiffs' general causation experts explained that dose is relevant and that they considered it. *See, for example*, Def. Ex. 1, Ritz Rpt. at 23 ("The lack of a safe level of lead in blood is ***not equivalent*** to the proposition that lead exposure will harm neurodevelopment ***at any level***. Rather, there is no threshold for exposure below which neurological harm does not manifest.") (emphasis added); *see also* Def. Ex. 4, Hu Rpt. At 36-37; Def. Ex. 7, Gardener Rpt. At 30-32, 79-80; Def. Ex. 10, Aschner Rpt. at 8-9, 18-19; Def. Ex. 13, Guilarte Rpt. at

8, 41-42.

b. Plaintiffs' Experts Did Not "Ignore" Dose or "Fail to Link" Dr. Jones's Calculations to the Literature

Plaintiffs' experts thoroughly considered dose and provided reliable bases linking Dr. Jones's computation of exposure from consumption of Defendants' baby foods to the doses observed in scientific literature as capable of causing harm. Defendants merely ignore Plaintiffs' experts' analyses and instead misquote the experts' deposition testimonies to argue that these experts "did not engage in any analysis to link the exposure levels calculated by Dr. Jones to the epidemiologic studies they claim support their opinions." Def. Br. 3 at 23. Defendants' argument is again meritless.

First, Defendants' claim that "Drs. Gardener, Shapiro, and Aschner failed specifically to address the issue of dose in their expert reports." *Id*. This is false. Significant portions of Drs. Gardener and Aschner's experts' reports are devoted to evaluating dose and the studies which identify specific doses of exposure associated with an increased risk of ASD/ADHD, which all three experts referred to when comparing the doses computed by Dr. Jones. *See* Def. Ex. 7, Gardener Rpt. at 84-85 (discussing studies identifying specific doses associated with an increased risk of ASD/ADHD and causal significance of doses from consumption of Defendants' baby foods); *see also id.* at 22-23, 30-32, 35-36, 47, 53, 55, 88-89, 111-114; Def. Ex. 10, Aschner Rpt. at 7-10, 16-19, 34-35, 55-56, 69, 71. It cannot be disputed that these experts evaluated and opined on the issue.[27]

Second, Defendants' assert that Dr. Hu "disclaimed" doing an analysis of dose and blood lead levels. Def. Br. 3 at 23. Again, untrue. Defendants cherry-pick a quote from Dr. Hu's deposition in support of their argument. However, Dr. Hu was merely asked "what range of changes in blood lead level do you consider to be meaningful or significant changes in terms of autism risk?" Def. Ex. 29, Hu Dep. at 231:4-6, which is a different issue to whether the doses of exposure from consumption of Defendants' baby foods are causally significant. On that point, Dr. Hu testified extensively:

> [A]s I opined in my report, lead exposure, even at very low levels in my review of the literature, can be expected to contribute to the risk of autism. There is no threshold that I'm aware of that's been identified below which, you know, lead

[27] As for Dr. Shapiro, his opinion is focused on the clinical presentation of ASD/ADHD, the recognized role of heavy metals in the etiology of ASD/ADHD as understood by clinicians treating children for these conditions, the genetics of both conditions, and the significance of exposure to heavy metals through food consumption. Def. Ex. 16, Shapiro Rpt. He is not offering a general causation opinion but is focused on biological plausibility.

> could not be expected to have a deleterious effect…the literature is simply straightforward in confirming the notion that lead exposure, ***including lead exposure at the levels encountered in children who would be eating contaminated baby food***, is likely to be a risk factor for autism… I have also seen data in the literature on the contribution of food to blood lead levels in children as young as zero to six months and twelve months to two years of age, in particular a publication by Zartarian et al…And that gave me a sense of how food at those ages, and presumably baby food, is a substantial contributor to blood lead levels that appear in children that young.

Id. at 83:3-9, 199:22-200:11, 233:7-234:2 (emphasis added); *see also* Def. Ex. 4, Hu Rpt. at 36-40. Defendants accuse Dr. Ritz of the same. *See* Def. Br. 3 at 23. However, again, Dr. Ritz's report contains an extensive discussion of the very issues that Defendants claim she ignored. *See* Def. Ex. 1, Ritz Rpt. at 7, 20, 23-25, 37-40, 46, 54, 71. And, Defendants omit the portion of Dr. Ritz' testimony where she reiterated her analysis. *See* Def. Ex. 26, Ritz Dep. at 163:2-10 ("**Q**. Can you identify any study on which you rely that finds an increased risk of ASD diagnosis at the mean lead or arsenic levels calculated by Dr. Jones? **A**. Well, you can calculate them from just about every study because, you know, assuming that there's a linear relationship, you just point out the points on the curve, and you have the relative risk."); *see also id*. at 156:4-11, 166:4-19, 181:16-182:3, 183:22-184:2. Lastly, Defendants criticize Dr. Guilarte for not identifying a "minimum dose and duration of exposure." Def. Br. 3 at 23. But, as explained by Plaintiffs' experts, the absence of a threshold dose does not preclude reaching a causal opinion between an exposure or outcome. *See e.g.* Def. Ex. 7, Gardener Rpt. at 31.

Third, Defendants make the argument that "Plaintiffs' experts just extrapolated," and assert that Dr. Ritz's only reference point was the recognition by the FDA and CDC that there is "no safe level" of exposure for lead. Def. Br. 3 at 23-24. This is a red herring. As explained above, Dr. Ritz identified specific epidemiological studies (*e.g.*, Kim, et al. (2016), Ha, et al. (2008), Tsuji, et al. 2015, etc.) as reference points for reaching an opinion regarding the causal significance of exposure from consumption of Defendants' products. In other words, the scientific consensus that there is "no safe level of exposure" to metals is merely a component of Dr. Ritz's opinions and does not mean that she did not compare the doses of exposure calculated by Dr. Jones to those reported in the epidemiological data (she did). *See* Def. Ex. 1, Ritz Rpt. at 70. Defendants also omit Dr. Hu's testimony (and ignore the unequivocal analysis in his report) that "the literature is simply

straightforward in confirming the notion that lead exposure, ***including lead exposure at the levels encountered in children who would be eating contaminated baby food***, is likely to be a risk factor for autism." Def. Ex. 29, Hu Dep. at 233:6-234:2 (emphasis added). Dr. Hu's opinions are by no means solely based on studies assessing the effects of specific doses of lead on cognition.

c. Defendants Misstate Plaintiffs' Experts' Opinions Regarding the Lack of a Safe Dose

Lacking a valid *Daubert* challenge to Plaintiffs' experts' opinions, Defendants instead invent opinions that Plaintiffs' experts are not offering. *See* Def. Br. 3 at 24-25. Defendants assert that "according to the experts' reasoning, eating only a single pouch of baby food containing trace levels of lead, or one carrot from the grocery store with detectable arsenic, could cause autism/ADHD." *Id*. at 24. ***Defendants do not cite any of Plaintiffs' experts' reports or depositions*** for this assertion beside an inapposite portion from the deposition of Dr. Shapiro. *See id.* But neither Dr. Shapiro, nor any of Plaintiffs' other experts, opine that eating one pouch of baby food or grocery store carrots causes ASD. Instead, Defendants plucked out a small portion of Dr. Shapiro's well-reasoned answer to fit their argument. But Dr. Shapiro was clear:

> **Q**. Do you have an opinion as to whether carrots, consumption of carrots, in higher or lower quantities is associated with an increased risk of autism or ADHD? **A**. [A]s I have said before in answer to slightly different questions, ***it would depend on the content of those carrots with respect to toxic heavy metals*** and the other things that the child is eating.

Def. Ex. 36, Shapiro Vol. 1 Dep. at 125:10-25 (emphasis added). Thus, Dr. Shapiro did not testify that eating one pouch of baby food or a single carrot causes ASD/ADHD, but rather that the causal significance of any particular exposure depends upon the dose and the unique facts of that case. *See id*. at 49:24-50:9, 51:2-20, 64:12-65:9, 71:11-24.

Next, Defendants conflate the scientific consensus that there is "no safe level of exposure" to metals with the spurious assertion that "any dose" can cause ASD/ADHD. Def. Br. 3 at 24-25. But, as Plaintiffs' experts make clear across their reports and depositions, these are distinct concepts:

> To begin, the concept of dose response is not synonymous with a threshold dose. And, when evaluating dose in the epidemiology of metals and neurodevelopmental conditions***, a threshold dose is inapplicable due to the stochastic nature of the outcome at issue***. As explained in other parts of this report, a threshold dose is the concept that there exists a defined dose of exposure to a toxin below which there is no risk of a specific outcome (here neurodevelopmental conditions symptoms

> diagnosed as ASD/ADHD) on a population level. However, the probability that a particular exposure will result in harm to an individual does not turn on whether that individual was exposed to a pre-defined threshold dose of the toxin. ***This is because manifestation of the outcome following exposure in any individual case is driven by the unique risk profile and circumstances of the exposed individual including the exposure timing during sensitive developmental periods and genetic vulnerability***. ***While the epidemiological literature has identified specific doses of metal exposure associated with an increased risk of developing ASD/ADHD (e.g., Kim et al. 2016)***, whether any individual child exposed to such doses will manifest symptoms diagnosed as ASD/ADHD is, inherently, probabilistic (i.e., a stochastic outcome)…The lack of a safe level of lead in blood is ***not equivalent*** to the proposition that lead exposure will harm neurodevelopment ***at any level***.

Def. Ex. 1, Ritz Rpt. at 38-39, 23 (emphasis added); *see also* Def. Ex. 7, Gardener Rpt. at 31. Accordingly, Plaintiffs' experts do not opine that ***any*** level of exposure ***will*** cause ASD/ADHD. Rather, while any amount of exposure confers a risk on a population level, the dose makes the poison ***depending on the individual***. Whether exposure ultimately results in a clinical presentation of symptoms significant enough to qualify for diagnosis depends on the unique biological profile of the exposed individual: "[T]here are…certain risk factors that have been identified that might increase a particular child's susceptibility to heavy metal exposure, methylation status, gene status…differences in gut microbiome or the integrity of the gut barrier. So there are many factors which one can identify which might contribute to increased risk of toxicity." Ex. 27, Shapiro Vol. 2 L.R. Dep. at 681:16-24, 674:21-25; *accord* Ex. 22, Shapiro Vol. 1 L.R. Dep. at 89:12-90:12, 173:16-174:3. Accordingly, Defendants' reference to case law deeming "no safe level" irrelevant is inapposite here because Plaintiffs' experts' general causation opinions are not predicated on ASD/ADHD being caused by "any" level of metal exposure.

3. Plaintiffs' Experts Correctly Considered and Analyzed Hundreds of Studies Linking Lead and Arsenic Exposure to an Increased Risk of ASD and/or ADHD

Defendants next mount a series of challenges to the underlying literature directly, arguing that the hundreds of published epidemiological studies supporting Plaintiffs' experts' general causation opinions are unreliable and unhelpful. *See* Def. Br. 3 at 25–46. Specifically, they argue that (a) studies looking at the symptomology of ASD and ADHD, (b) studies involving children outside the United States, (c) studies looking at prenatal and older children, and (d) studies looking at cross-sectional data are unreliable and cannot be used to support a general causation opinion. By slicing

away various types of peer-reviewed studies, Defendants hope to erase the overwhelming body of scientific data clearly linking lead and arsenic exposure to ASD and/or ADHD. This is, of course, not how science is done. Experts must carefully consider all data and weigh that data against its strengths and weaknesses. *See, e.g.*, Ex. 24, Ritz Vol. 1 N.C. Dep. at 277:18–278:13 ("It's very important that you actually have studies with different designs to compare."); *see also In re Bextra and Celebrex Mktg. Sales Practices and Prods. Liab. Litig.*, 524 F. Supp. 2d 1166, 1176 (N.D. Cal. 2007) ("ignoring the great weight of the evidence that contradicts [a preferred] conclusion . . . does not reflect scientific knowledge, is not derived by the scientific method, and is not 'good science'"). The studies Defendants seek to exclude each provide important insight into the causal question—each being the subject of peer-reviewed research by independent scientists exploring the ability of lead and arsenic to increase the risk of ASD and/or ADHD in children. While no study is perfect, when hundreds of different researchers, using different methodologies, in different populations, and different ways of measuring exposure converge on the same truth, it provides an answer to the question at the heart of this general causation inquiry, as Dr. Hill put it: "is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?" Ex. 78, Hill (1965) at 299; *see* Def. Ex. 33, Gardener Dep. at 254:23–255:1 ("One of the great things about this literature and one of the strengths is that there was a confluence of data from many different populations of kids.").

a. Studies that Consider ASD and/or ADHD Symptoms Are Relevant and Helpful

Defendants argue that Plaintiffs' experts' opinions are "unreliable" because their review of the literature included studies assessing the relationship between exposure to heavy metals and symptoms of ASD/ADHD. *See* Def. Br. 3 at 27-31. As an initial matter, this exact argument was repeatedly made, and lost, in the state court litigation. As Judge Hogue noted in the *NC* case, "it is not unreasonable to measure the presence and severity of ASD's characteristic behavioral disorders to approximate diagnosed ASD. Where on the spectrum the presence and severity of various behaviors becomes diagnosable as ASD is a matter of degree, and reasonable clinicians can disagree about where on the spectrum as ASD diagnosis is appropriate." *NC*, 2022 WL 21778549, at *14. And this makes sense. ASD and ADHD are, by definition, "behavioral" or "symptom-based" diagnoses,

which fall on a spectrum. *See* Def. Ex. 16, Shapiro Rpt. at 8; Ex. 22, Shapiro Vol. 1 L.R. Dep. at 86:11-87:25. Reliance on studies that evaluate the relationship between metal exposure and ASD/ADHD symptoms is *entirely* appropriate. In fact, studies assessing symptoms of ASD/ADHD from metal exposure "are often a ***more useful end point*** than diagnosis in part because they indicate the degree of impairment that a person has and also are more sensitive to the existence of a spectrum of severity in the population." *Id.* at 199:9-21 (emphasis added). As Dr. Ritz explains:

> I do not limit my analysis to the diagnoses, as a large number of neurodevelopmental milestones for behavior(s) (such as in social communication or eye tracking) as well as symptom(s) associated with ASD and/or ADHD are considered prodromal or phenotypic markers of the development of ASD and/or ADHD that can occur before clinicians are able to make a formal diagnosis, ***which is why it is important to consider study endpoints other than diagnoses***…there are various relevant endpoints on a spectrum of severity (hence autism 'spectrum' disorder) and not every child exhibits every behavior or symptom found in children with ASD and/or ADHD and some may be considered 'attenuated phenotypes' or 'autistic traits' that are generally assessed in a quantitative manner and are less specific than the core categories of ASD/ADHD but are nevertheless ***useful tools for screening and diagnosing purposes as well as research***.

Def. Ex. 1, Ritz Rpt. at 19 (emphasis added); *accord* Def. Ex. 29, Hu Dep. at 131:20-132:3; Def. Ex. 7, Gardner Rpt. at 21 ("[I]t is important to consider that ASD and ADHD are "spectrum" disorders, meaning that they are composed of a variety of behavioral manifestations and symptoms as well as varying degrees of severity. As such, a binary interpretation of the epidemiological literature on heavy metals and ASD/ADHD is not appropriate, i.e., limiting the inquiry to whether cases have a 'formal' ASD/ADHD diagnosis or not."). Considering studies that evaluated symptoms as well as strict diagnosis is, simply put, good science.

That said, Plaintiffs' experts did not, as Defendants suggest, limit their evaluation to *only* studies assessing symptoms. Plaintiffs' experts also relied upon a vast body of literature that observed associations between metal exposure and diagnosed ASD/ADHD. *See* Def. Ex. 1, Ritz Rpt. at 43-67; *accord* Def. Ex. 7, Gardener Rpt. at 16 and 38-79; Def. Ex. 10, Aschner Rpt. at 26-65; Def. Ex. 4, Hu Rpt. at 13; Def. Ex. 13, Guilarte Rpt. at 19-31; Def. Ex. 16, Shapiro Rpt. at 34-35; Ex. 22, Shapiro Vol. 1 L.R. Dep. at 89:18-20, 94:3-8.

Defendants also criticize Plaintiffs' experts for relying on studies that evaluated the association between metal exposure and neurodevelopmental issues with ASD screening

questionnaires. *See* Def. Br. 3 at 29-30. But such an argument flies in the face of basic neuroscience. As Plaintiffs' experts explained, the fact that studies such as Kim, et al. (2016) and Alampi, et al. (2021) observed a strong association between early life metal exposure and higher scores on the Autism Spectrum Screening Questionnaire ("ASSQ") and Social Responsiveness Scale ("SRS") lends greater support to the causal role of metals in ASD etiology:

> [The study children] had ASD-like features and ASD is a spectrum disease. And, yes, we screen with these screeners for ASD. If we are nonspecific, which we are in these screeners because we have high sensitivity, the likelihood of finding an effect is very low. So them seeing an effect is actually extremely surprising…This study…uses a screener with approved questions and validated questions that relate to ASD…But the only effect you usually see when you are more specific is that effects get stronger, not weaker…it actually means that kids who otherwise are okay are harmed, as well, and are harmed in exactly the same way that the kids who get an ASD diagnosis are harmed.

Ex. 24, Ritz Vol. 1 N.C. Dep. at 231:8-232:12; *see also* Def. Ex. 29, Hu Dep. at 273:8-15 ("[B]oth of these instruments are very highly correlated with and predictive of the diagnosis of ASD. There's a pretty robust literature about that. And as such, I think they have direct relevance to the question of the etiology of ASD.") (emphasis added); Def. Ex. 51, Shapiro Vol. 1 N.C. Dep. at 144:5-15; Ex. 89, Gardener Vol. 1 N.C. Dep. at 362:20-363:2.

Next, Defendants assert that "Plaintiffs' experts did not conduct any analysis to determine whether the purported impact of heavy metal exposure varied depending on the particular outcome studied—*e.g.*, diagnosed autism vs. 'autistic behaviors'—in terms of the strength of any supposed association" and "did no independent analysis of whether the particular behavior(s) or symptom(s) studied are correlated with autism or ADHD—as opposed to some other neurological or cognitive endpoint not at issue in this litigation." Def. Br. 3 at 30-31. This is a red herring. As Dr. Ritz and Gardener explained, the fact that studies assessing "behaviors" observed associations between metal exposure and symptoms of ASD/ADHD underscores the strength of the association between metal exposure and "diagnosed" ASD/ADHD because "the likelihood of finding an effect" using screening tools is "very low" due to non-differential misclassification when utilizing screening tools. Ex. 24, Ritz Vol. 1 N.C. Dep. at 231:8-232:12; *see also* Ex. 89, Gardener Vol. 1 N.C. Dep. at 362:20-363:2 ("That would be considered a non-differential misclassification of the outcome, and I view that as a strength because when you see associations when the outcome is classified in different ways, you

have more confidence that your association is actually there, that it's causal, that it's real, that it's not spurious based on sources of bias."). Moreover, Defendants are arguing semantics. As Dr. Shapiro explained in his report:

> Symptoms that contribute clinically to the diagnosis of ASD and ADHD can emerge as a result of perturbations to the developing nervous system caused by environmental exposure, such as by heavy metals. Neurodevelopmental toxicity simply refers to systemic interference with neurodevelopment caused by metal exposure that can manifest in a variety of symptoms …

Def. Ex. 16, Shapiro Rpt. at 35. As Dr. Shapiro further testified:

> Q. There's a difference, right, Doctor, between autism as a clinical end point and an individual symptom that may or may not meet criteria for autism. You appreciate that concept? A. So in a clinical context, I think those things are inseparable. Semantically…is there a difference? There is, but…the difference is not of great clinical relevance. What we're interested in when assessing a child from a clinical standpoint, as a neurologist or psychiatrist or pediatrician or what have you, is what are their symptoms, not what is the label…

Ex. 22, Shapiro Vol. 1 L.R. Dep. at 165:11-24 (emphasis added); *see also* Ex. 23, Shapiro Vol. 1 N.C. Dep. at 84:23-85:8; Def. Ex. 46, Gardener L.R. Dep. at 81:23-82:3. Accordingly, there is a sound methodological—indeed, common sense—basis for relying on studies assessing the adverse effects of metals on ASD/ADHD behavior in considering the causal significance of metal exposure in the etiology of ASD/ADHD.

b. Plaintiffs' Experts Appropriately Considered Studies Involving Children Outside the United States

Defendants next argue that reliance on epidemiological studies involving children outside the United States is inappropriate because children outside the U.S. generally have higher metal exposures. *See* Def. Br. 3 at 36–39. But this is decidedly not how science works.

First, it should go without saying, but "lead is a neurotoxin no matter where you are." Def. Ex. 26, Ritz Dep. at 140:25–141:5. The ability of lead and arsenic to penetrate the BBB of an infant and interfere with neurodevelopment is not constrained by geography. Babies in India are biologically the same as babies in the United States. Getting data from different geographical sources is critical in assessing causation:

> Q. As an epidemiologist working in environmental epidemiology, is it scientifically appropriate, when you're trying to assess whether heavy metal exposures from arsenic and lead can cause ASD or ADHD, to limit your review of that

> epidemiological literature to specific geographic regions? ***A. Absolutely not.*** Q. Why? A. Because you want to do what we call triangulation, meaning we want heterogeneous studies showing us what the possibilities of the associations look like under different conditions. Environmental epi is a very hard business because most of the time, the environmental exposures are lower … so you need very large sample size and very good exposure assessment to actually see anything. Q. Is it scientifically appropriate to look at the results of epidemiological studies from various geographic regions in weighing and assessing the causation question? A. ***Absolutely. That's what you should be doing***.

Def. Ex. 43, Ritz L.R. Dep at 316:19–317:16 (emphasis added). Dr. Gardener explains:

> [T]he beauty of sort of looking at different time periods and kids from different socioeconomic status groups, kids from different parts of the United States and from different countries, is that when you see associations consistent in different study populations and over different time periods, that is actually reassuring even if the average level -- the average blood lead level in a child in 1995 in Egypt might be quite far off from a child in this study.

Def. Ex. 33, Gardener Dep. at 255:9–19; *accord* Def. Ex. 34, Aschner Dep. at 244:8–246:5.

To be clear, Plaintiffs' experts are not the only researchers who combine the results of the U.S. and foreign studies; the numerous systematic reviews and meta-analyses by independent researchers—all subject to considerable independent peer-review—also considered U.S. and foreign studies in combination. *See* Def. Ex. 29, Hu Dep. at 102:1–103:3; 110:20–111:16. Plaintiffs' experts' decision to consider foreign studies is, more likely than not, reliable considering it is exactly what other independent scientists do. Defendants' attacks on Plaintiffs' experts' interpretations and applications of these meta-analyses—Stojsavljevic et al. (2023), Ding et al. (2023), Shiani et al. (2023), and Nakhaee et al. (2022)—ignore important aspects of those studies. For example, Defendants attack Stojsavljevic et al. (2023)'s overall conclusions as reflecting studies involving children with higher overall lead burdens. *See* Def. Br. 3 at 36-37. But Defendants ignore the authors' conclusions that "[m]ost studies reported higher levels of Pb in the hair of case children than in controls, in Saudi Arabia, Oman, Iraq, . . ., ***[and] the USA***." Def. Ex. 88, Stojsavljevic (2023) at 13 (emphasis added). Likewise, "[m]ost studies reported higher Pb levels in the whole blood of autistic children than in neurotypical controls, ***including in the USA***." *Id.* at 14 (emphasis added). Similarly, the Ding et al. (2023) authors found that "ASD patients in ***North America*** . . ., Asia . . ., and Europe . . . had higher [lead] concentration than the healthy controls in these regions." Def. Ex. 64, Ding, et al. (2023) at 3 (emphasis added). Defendants ignore the fact that both Shiani et al.

(2023) and Nakhaee et al. (2022) noted that Adams et al. (2013)—a U.S. Study— found higher levels of blood and urinary lead among ASD cases than controls. Def. Ex. 65, Shiani, et al. (2023) at 7; *see also* Def. Ex. 75, Nakhaee, et al. (2022) at 1-6.

It is a basic feature of observational epidemiology that researchers focus on populations with greater exposures. Indeed, when an outcome is rare, epidemiologists deliberately consider locations where exposure is higher because it facilitates assessing dose-response. *See* Def. Ex. 26, Ritz Dep. at 139:19–141:5 ("Well, there are of studies from high exposure countries because the exposures are of particular concern, and it's also easier to study the outcome when the exposure is higher."); *see also id.* at 141:7–22 (explaining how U.S. funds international studies because it helps identify exposure outcomes applicable to the U.S. population).

Second, Defendants' contention that Plaintiffs' experts rely exclusively on studies conducted outside the United States simply is not true. Plaintiffs' experts cite multiple epidemiological studies conducted in the U.S. that show associations between heavy metal exposures and ASD/ADHD. For example, Drs. Hu, Guilarte, and Shapiro discuss Adams et al. (2013), an observational study of American children, finding that "children with autism have higher average levels of several toxic metals," and that ASD cases "had higher levels of lead in [red blood cells] and higher urinary levels of lead." Ex. 90, Adams, et al. (2013) at 171; *see also* Def. Ex. 4, Hu Rpt. at 39; Def. Ex. 13, Guilarte Rpt. at 19; Def. Ex. 16, Shapiro Rpt. at 29. Likewise, all of Plaintiffs' experts, except Dr. Shapiro, discuss Ji et al. (2018), in which the authors found evidence of an association between lead exposure and ADHD amongst a study population from the Boston Birth Cohort. *See* Def. Ex. 91, Ji et al., (2018) at 1; *see also* Def. Ex. 1, Ritz Rpt. at 65; Def. Ex. 7, Gardener Rpt. at 23; Def. Ex. 4, Hu Rpt. at 13, 35; Def. Ex. 13, Guilarte Rpt. at 25-26; Def. Ex. 10, Aschner Rpt. at 50. Additional examples abound. Chiodo et al. (2007) found an association between lead exposure and ADHD amongst children in Michigan. *See* Def. Ex. 1, Ritz Rpt. at 65 (discussing the study); Ex. 91, Chiodo et al., (2007) at 539 (noting study population). Roberts, et al. (2013) found associations between lead and ASD among a nationwide American study population. *See* Def. Ex. 7, Gardener Rpt. at 50-51; Ex. 92, Roberts, et al., (2013) at 978. In his report, Dr. Hu discusses a 2022 meta-analysis conducted by Dalla et al. in which the authors identified three North American studies – Boucher et al. (2012)

(Canadian data), Arbuckle et al. (2016) (Canadian data), and Ji et al. (2018) (American data) – that satisfied the meta-analysis's strict inclusion criteria and found associations between lead exposure and ADHD. *See* Def. Ex. 4, Hu Rpt. at 167; *see also* Ex. 93, Dalla, et al., (2022) at 4-6.

Finally, this attack on foreign studies makes no logical sense. In observations epidemiology, the focus is on *relative* risk—how the exposed group compares to the unexposed group (or, in the context of lead and arsenic, how the greater exposed group compares to the lesser exposed group). The foreign studies, just like U.S. studies, compare exposures within the *same* population and, from that population, compare the *relative* differences in disease outcome. So, if the overall baseline exposure is higher in one country, it will be higher in both groups—the study, thus, reflects whether differences of metal exposure within comparable groups increases the risk of a disease outcome. It is relative to the population studied. This is why these studies can provide valuable insight on whether additional exposures, relative to the control group, cause ASD and/or ADHD.

c. Plaintiffs' Experts Appropriately Considered Prenatal Exposure Studies as Part of their Systematic Review of the Available Literature

Defendants contend that Plaintiffs' experts should not have considered or relied on studies examining heavy metal exposure during prenatal periods because those populations do not consume baby food. *See* Def. Br. 3 at 33–35. To be clear, Plaintiffs' experts considered scores of postnatal studies, including studies that specifically looked at children in their early developmental period. Defendants' argument, however, is that Plaintiffs' experts should not have considered any prenatal data in assessing causation. *See id.*

Defendants begin their argument claiming that the prenatal data does not support causation. *See id.* at 31 ("[T]hose studies largely fail to show *any* positive association[.]"). This is not correct. In Skogheim (2021), the authors looked at prenatal exposures and concluded: "Results from the present study show several associations between levels of metals and elements during gestation and ASD and ADHD in children. The most notable ones involved arsenic … and lead. Our results suggest that even population levels of these compounds may have negative impacts on neurodevelopment."). Def. Ex. 77, Skogheim (2021) at 1. Alampi (2021) observed that "[h]igher gestational levels of lead… were associated with higher mean SRS scores."). Def. Ex. 90, Alampi (2021) at *1807. In

Tsai (2023), they observed that "[m]aternal urinary concentrations of … Pb were also positively associated with the odds of … autism spectrum problems" and that "prenatal exposure to metal mixtures … had significantly positive associations with the increased odds of autism spectrum problems in children." Ex. 100, Tsai (2023) at 5. In Long (2019), which had a very small number of participants (37 cases), "[t]he association between arsenic and ASD was shy of statistical significance with an adjusted OR per 1 mg/L increase in arsenic of 1.50 (95% CI: 0.92–2.42), but the results did suggest a contribution of prenatal arsenic exposure in relation to ASD." Def. Ex. 66, Long, et al. (2019); *see also* Def. Ex. 7, Gardener Rpt. at 60. Neugebauer (2015) observed increased ADHD behavior with increased prenatal lead exposure. *See* Ex. 101, Neugebauer (2015) at *153. The assertion that prenatal data does not support causation is demonstrably false.

That said, there is nothing improper, scientifically, with considering prenatal data. *E.g.*, *Junk v. Terminix Int'l Co.*, 577 F. Supp. 2d 1086, 1092–93 (S.D. Iowa 2008) (admitting general causation testimony about chemical's ability to cause neurodevelopmental harm based on prenatal and postnatal studies). These experts considered the prenatal data to help better consider biological plausibility and reverse causality. As Dr. Gardener explains:

> For the same reason, I included in my literature review studies on prenatal exposure to heavy metals. The prenatal period represented a different etiological period from the one in question in this legal matter. However, I found this literature highly informative in this case because it also allayed my concerns about temporality and reverse causation, in addition to helping me understand biological plausibility.

Def. Ex. 7, Gardener Rpt. at 22. Neurodevelopment does not begin and end within the narrow window when children consume baby food. Brain development is protracted and continuous and "occurs during the prenatal period" and "continues on after" for "years and years." Def. Ex. 33, Gardener Dep. at 140:18-141:8, 188:23-189:16; *see also* Def. Ex. 16, Shapiro Rpt. at 16 (describing prenatal and postnatal neuronal development). Dr. Aschner emphasizes this point: "Brain development is a protracted process that starts about 2 weeks after conception and comprises several key stages that progress through the neonatal and infant period well into adolescence before the brain is fully mature." Def. Ex. 10, Aschner Rpt. at 58. Thus, the "consideration of the potential effects of arsenic, and lead on brain development in the early years of childhood is *only* complete if one considers the origins of this process during the prenatal months." *Id.* (emphasis added). And this

makes sense. If heavy metals damage the developing brain *prenatally*, they can damage it *postnatally*. As Dr. Ritz explains, prenatal neurotoxicity "gives you a good hint at what this agent can actually do" and is "one of the arguments for neurotoxicity in general." Def. Ex. 26, Ritz Dep. 41:13-25. The prenatal studies are also helpful in considering the potential for reverse causation in other studies. *See* Def. Ex. 1, Ritz Rpt. at 13 ("[Studies conducted on prenatal and early life exposures… refute the likelihood of reverse causation."); *see also* Def. Ex. 7, Gardener Rpt. at 22. Again, if one sees the impact of lead and arsenic on later development of ASD and/or ADHD, it assuages any epistemological concerns about whether exposures preceded the outcomes.

None of Plaintiffs' experts exclusively relied on prenatal data; but they all did consider it as part of their overall evaluation of the scientific literature. Through consideration of all scientific data, especially data that can help assess biological plausibility and risk of reverse causation, is the hallmark of reliability. Defendants' concern about consideration of prenatal data is without merit.

4. Plaintiffs' Experts Appropriately Considered Cross-Sectional Studies and Carefully Weighed Concerns Related to Temporality

a. Cross-Sectional Studies Provide Valuable Evidence

Defendants argue that Plaintiffs' experts incorrectly relied on studies (cross-sectional data) that, according to them, "cannot establish the required temporal sequence between exposure and outcome[.]" Def. Br. 3 at 41; *see also id* at 39-47. This is an argument that Defendants have repeatedly made and lost in state court. *E.g.*, *NC*, 2022 WL 21778549, at *8 ("Drs. Ritz's and Gardener's opinions on temporality are sufficiently logical and non-speculative."). Defendants' renewed effort to ignore large swaths of peer-reviewed literature remains unpersuasive.

In assessing whether exposure to neurotoxic heavy metals in early life can cause ASD/ADHD, Plaintiffs' experts carefully weighed evidence from several different types of studies, namely prenatal studies, postnatal prospective cohort studies, case-control studies, cross-sectional studies, and numerous meta-analyses. With respect to the cross-sectional data, Plaintiffs' experts specifically considered temporality and provided exhaustive, reasoned analyses of: (1) the limitations of cross-sectional data in light of concerns with temporality; (2) why temporality and the related issue of reverse causation do not explain the consistent associations observed in the data (with reference, *inter alia*, to prospective and prenatal data, and the utility of various biomarkers reflecting long-term

exposure); and (3) the bases for their reliance, at least in part, on cross sectional data. *See* Def. Ex. 7, Gardener Rpt. at 15, 18-24, 27, 47, 49-50, 56, 60, 63, 69-70, 77; Def. Ex. 1, Ritz Rpt. at 13, 19-22, 37-38, 46, 55, 57, 61, 63, 65, 68, 70; *see also* Def. Ex. 10, Aschner Rpt. at 54-57; Def. Ex. 4, Hu Rpt. at 11, 19-20, 28, 32-34; Def. Ex. 35, Guilarte Dep. at 63:13-64:9, 112:17-22, 113:19-114:10. As Plaintiffs' experts explain, concerns with temporality are mitigated by multiple lines of evidence.

First, even in cross-sectional studies, it is possible to establish a temporal relationship between exposure and disease by utilizing biomarkers such as blood, urine, hair, teeth, bone, toenails, and tissue, which permit reliable inferences regarding exposures *predating* disease onset. *See* Ex. 24, Ritz Vol. 1 N.C. Dep. at 38:17-21, 39:14-23, 40:24-41:13, 41:23-42:1, 43:10-18, 198:4-200:16; *see also* Ex. 36, Reference Manual at 586, fn. 109 ("biomarkers are likely to be increasingly relied on to demonstrate exposure"). As Dr. Hu explained: "[O]ne of the interesting features of biomarkers is that -- particularly biomarkers that represent cumulative exposure, have a time referent ***that is not simply acute exposure***. It actually summarizes exposures that ***occur previously***." Def. Ex. 29, Hu Dep. at 260:23-261:6. He cautions that "this temporality condition has to be interpreted carefully. It doesn't necessarily mean, for instance, that a cross-sectional study has no relevance to the issue of temporality." *Id.* For example, "the general toxicokinetics of lead is that once it's in blood, there is a proportion of lead that then goes into bone. This is a topic that my research group has studied in detail. And…***those bone lead stores are not inert***. They actually leak out later in life. So a blood lead level taken at seven years of age is probably ***likely to include whatever lead exposure they had from much earlier ages***[.]" *Id.* at 74:2–14; *see also id.* at 122:10–22; Ex. 24, Ritz Vol. 1 N.C. Dep. at 38:17-21, 41:23-42:1, 39:14-23 ("That's actually the incredible strength of these types of studies…It can go quite far back because lead is stored in the bone and there's a constant replacement of lead in the blood from the bone … that's an advantage of biomarker studies over other studies"); *see also* Ex. 105, Aschner Vol. 1 N.C. Dep. at 115:10-158:4 (discussing reliability of biomarkers). Accordingly, the fact that cross-sectional studies consistently report differences in metal content between ASD and non-ASD controls based on reliable biomarkers that ***capture pre-disease*** exposures fits reliably within the consistent associations observed in the prospective studies.

Second, the existence of other data ameliorates concerns with the temporality limitation of the

cross-sectional studies. *See* Def. Ex. 7, Gardener Rpt. at 19 ("If the prospective data is consistent with the data observed in retrospective and cross-sectional studies, then … concerns about temporality in cross-sectional studies are ameliorated."); *see also id.* at 20 ("[T]he availability of prospective studies, data on prenatal exposure, studies on early life (pre-diagnosis) exposure to heavy metals via analysis of baby teeth, and translational research all demonstrate the etiological relevance of heavy metal exposure ***prior*** to manifestation of disease.") (emphasis in original); *accord* Ex. 24, Ritz Vol. 1 N.C. Dep. at 64:8-65:2; Def. Ex. 10, Aschner Rpt. at 54; Def. Ex. 11, Ritz Rpt. at 45-46. One such study is Kim (2016), a large study in school age children that measured blood lead in 7–8-year-old children prior to assessing autistic behavior 4-5 years later. As Dr. Ritz noted, "[t]his study is particularly important as it establishes temporality between exposures and outcomes and has a large enough size to allow investigating associations with relatively low blood lead levels." Def. Ex. 1, Ritz Rpt. at 46; *accord* Def. Ex. 4, Hu Rpt. at 28-30; Def. Ex. 7, Gardener Rpt. at 22-23; Def. Ex. 10, Aschner Rpt. at 34-35. In addition to prospective cohort studies like Kim (2016), Plaintiffs' experts also relied upon several other studies, including a twin study (Arora 2017) which utilized a biomarker that reflects lead exposure specific to the early infancy period; thus also providing clear evidence of exposure preceding the development of ASD in a specific etiological period. *See* Def. Ex. 68, Arora, et al. (2017); *see also* Def. Ex. 4, Hu Rpt. at 32-33; Def. Ex. 1, Ritz Rpt. at 45; Def. Ex. 7, Gardener Rpt. at 51. Dr. Hu explained that Arora (2017) "provides strong support for fulfilment of the Bradford Hill criterion of temporality, which, in turn, is a critical dimension of the assessment of the causal relationship between lead exposure and ASD may have behaviors that increase their exposure to lead in their environment." Def. Ex. 4, Hu Rpt. at 32.

b. Defendants' Attempt to Impugn an Entire Swath of Peer-Reviewed Literature Is Little More Than Speculation

First, Defendants assert that certain behaviors exhibited by some autistic children—such as compulsive hand-to-mouth behavior known as "pica"—can increase their exposure to heavy metals and "thereby raise the possibility of reverse causation[.]" Def. Br. 3 at 40-41. But this speculative "worry" is just that—speculation. As an initial matter, Defendants lost this very argument in state court on multiple occasions. *See NC*, 2022 WL 21778549, at *7–9. The same reasons militate denying Defendants' challenge here. As explained above, the use of biomarkers in cross-sectional

studies (permitting reliable inferences regarding pre-diagnosis exposures), ***and*** the availability of prospective studies (which, by definition, assess exposure prior to disease) make it highly unlikely that the consistently positive results observed across multiple studies employing varied designs and conducted across the globe are due to reverse causality. *See, e.g.,* Ex. 7, Gardener Rpt. at 19-20; *see also* Ex. 89, Gardener Vol. 1 N.C. Dep. at 363:3-14; Def. Ex. 1, Ritz Rpt. at 13, 65; Def. Ex. 24, Ritz Vol. 1 N.C. Dep. at 88:19-90:23, 198:10-200:16; Def. Ex. 10, Aschner Rpt. at 53-65. Reliable biomarker data and prospective studies do not suggest, whatsoever, that reverse causality is leading to falsely positive cross-sectional studies. Indeed, the notion that ASD children with pica ingest more lead and arsenic is not supported by the data. For lead, researchers examined whether ASD children with pica ingest more lead than other ASD children without pica, and they found *no difference* in BLLs. *See* Ex. 106, Brown (2025) at 1. In another study examining the lead and arsenic levels of ASD children in Arizona, the researchers observed no difference between in lead between ASD children with PICA and ASD children without pica (0.65 v. 0.60) and statistically significant more arsenic in the *ASD children without pica* compared to ASD children with pica. *See* Ex. 107, Adams (2006) at 198, Tbl.2. These studies lay bare the speculative notion that ASD children have high lead and arsenic levels because of pica. This is particularly true for arsenic. While lead, especially in older homes, can be found in the environment (mostly from lead-based paint), there is absolutely no evidence that arsenic contaminates objects in a child's immediate environment that they would be likely to put in their mouths. *See* Def. Ex. 7, Gardener Rpt. at 23-24; *see also* Ex. 24, Ritz Vol. 1 N.C. Dep. at 68:18-69:24, 70:15-21, 322:6-323:16. Thus, while the pica argument, on its surface, might have some appeal, it finds no support in the data. It amounts to self-serving speculation.

Second, Defendants speculate that "[r]estrictive eating behaviors among children with autism can also lead to nutrient deficiencies that, in turn, may lead to increased absorption of heavy metals." Def. Br. 3 at 41. However, as Plaintiffs' experts explained, Defendants' criticism is overly broad and misleading. *See* Def. Ex. 26, Ritz Dep. at 277:6-12; *see also id*. at 279:24-280:17. Defendants' cherry-picked citation to Dr. Guilarte's deposition does not help, as Dr. Guilarte clarified that whether deficiencies can explain the higher levels of metals in ASD subjects depends, *inter alia*, on "on the level of exposure of intake." Def. Ex. 35, Guilarte Dep. at 192:10-17.

Third, Defendants argue that that just because Plaintiffs' experts relied, in part, on cross-sectional studies for their opinions, that somehow means that they did not consider temporality or whether temporality was met in the cross-sectional data. Def. Br. 3 at 41-42. This ignores Plaintiffs' experts' extensive consideration of the issue. For example, Defendants cite Dr. Hu for the proposition that temporality is an issue in cross-sectional data but omit his exhaustive explanation of why temporality is still satisfied. *See* Def. Ex. 29, Hu Dep. at 61:18-62:18, 73:20-74:16, 260:23-261:6; *see also* Ex. 24, Ritz N.C. Vol. 1 Dep. at 38:17-21, 39:14-23, 41:23-42:1. Defendants also accuse Dr. Aschner of improperly considering temporality but ignore the multiple pages of his report devoted to the issue as well as his clear deposition testimony. *See* Def. Ex. 10, Aschner Rpt. at 53-65; *see also* Def. Ex. 34, Aschner Dep. at 236:12-21, 264:6-18.

Fourth, Defendants assert that "Plaintiffs' experts did not attempt to evaluate the impact of nutrient deficiencies in children with autism on levels of lead or arsenic in biomarkers." Def. Br. 3 at 42. This is misleading. Defendants cite the testimonies of Drs. Ritz and Aschner in support of their argument but omit their complete responses. Dr. Ritz clearly explained that she considered the issue, emphasizing that the effects depend on multiple factors including socioeconomic status, geographic location, and individual family circumstances, and provided her reasons for why it does not explain the consistent causal trend observed in the literature between exposure to heavy metals and ASD/ADHD. *See* Def. Ex. 26, Ritz Dep. at 90:13-91:9, 277:6-280:17. As for Dr. Aschner, Defendants ignore his extensive discussion of reverse causation in his report when he provides multiple lines of evidence for why the behavioral traits of ASD do not explain the consistent findings of the studies assessing metal exposure and ASD. *See* Def. Ex. 10, Aschner Rpt. at 53-55.

Fifth, Defendants claim that Plaintiffs' experts had no coherent response when asked about reverse causation but, tellingly, do not quote any of Plaintiffs' experts. That is because Plaintiffs' experts' testimony undercuts Defendants' argument. *See, e.g.*, Def. Ex. 26, Ritz Dep. at 57:22-59:23.

c. The Vast Body of Data Considered by Plaintiffs' Experts Supports Causation

Defendants contend that even the studies that satisfy temporality do not support Plaintiffs' experts' opinion. *See* Def. Br. 3 at 43-47. This is a meritless argument that Defendants have roundly lost in state court. Defendants' tactic has not since changed: atomize the data by excluding studies

based on criteria lacking sound scientific basis to fit Defendants' *a priori* position of no causation.

First, Defendants claim that "there are simply no studies evaluating postnatal exposure to arsenic and autism that satisfy temporality." Def. Br. 3 at 43. This is a red herring. As explained by Plaintiffs' experts, "[a]ssociations observed between maternal arsenic levels during pregnancy and an increased risk of ASD following birth supports the understanding that arsenic levels early in life, ***prior to an ASD diagnosis***, are in fact etiologically relevant, rather than increased arsenic levels being observed as a consequence of ASD." Def. Ex. 7, Gardener Rpt. at 60. Sound scientific practice does not willfully ignore data. Rather, the totality of the available evidence (such as prenatal data) supports Plaintiffs' experts' reliance on postnatal and cross-sectional studies of arsenic.

Second, contrary to Defendants' assertion, the 3 prenatal studies on arsenic and ASD cited by Defendants, Def. Br. 3 at 43, *all* support Plaintiffs' experts' opinions because all four observed an association between prenatal arsenic exposure and ASD. *See* Def. Ex. 77, Skogheim (2021) at 1 ("Results from the present study show…notable [associations]…between levels of arsenic…and lead…during gestation and ASD and ADHD in children."); *see also* Def. Ex. 66, Long, et al. (2019) at 16 ("The present study showed that environmental…metals, and their biological activities can…modify ASD risk by influencing the hormone receptor function."); Def. Ex. 71, Dou (2024) at 11 ("This study suggests that prenatal exposure to toxic metals…is associated with risk of ASD or non-typical development in offspring."). As Plaintiffs' experts explained (and state court judges previously found), the lack of statistical significance does not preclude a causal conclusion. *See* Ex. 89, Gardener Vol. 1 N.C. Dep. at 209:9-210:16; *see also NC*, 2022 WL 21778549, at *8. And, with respect to Skogheim, Defendants' atomization is meritless as Dr. Ritz clearly explained how the non-linear association in that study clearly supports causation. *See* Ex. 24, Ritz Vol. 1 N.C. Dep. at 136:10-154:23.

Third, Defendants' rote attack on the Arora (2017) has also been repeatedly made and lost. *See* Def. Br. 3 at 44. Defendants claim that infants do not eat baby food during one of the windows of time in Arora which observed an association between lead exposure and ASD. But this misses the point. Plaintiffs' experts' reliance on this study is in support of the causal evidence between postnatal lead exposure and ASD in a study that clearly satisfies temporality. *See* Def. Ex. 7, Gardener Rpt. at

51 (the study "provided important insight about the ability of lead exposure during infancy to cause ASD"). Just because the source of exposure is not identified in the study does not render it unreliable. Also, it is factually inaccurate that the study did not observe an association "for any other postnatal window". Def. Br. 3 at 44. As Dr. Ritz explained: "they probably didn't have enough statistical power to show the other effects… ***But when you look at the curves, you see there is still effect***." Ex. 24, Ritz Vol 1 N.C. Dep. at 213:7-214:11 (emphasis added).

Fourth, Defendants invoke four prenatal lead exposure studies and dismiss all but one for lack of statistical significance ***notwithstanding a positive association being observed across all four studies***. *See* Def. Br. 3 at 44. But as Plaintiffs' experts explained, it is improper to discount studies observing an association between exposure and outcome merely because of the lack of statistical significance. *See* Ex. 89, Gardener Vol. 1 N.C. Dep. at 209:9-210:16. Defendants reference Dr. Guilarte's testimony for the proposition that the Dou (2024) is unreliable merely because the study did not adjust for "multiple testing". Def. Br. 3 at 45. Putting aside the fact that such criticisms go to weight, not admissibility, Dr. Guilarte never faulted Duo (2024) for not adjusting. *See* Def. Ex. 35, Guilarte Dep. at 74:10-75:1. The portion of Dr. Guilarte's deposition cited by Defendants was not even discussing the Dou (2024) study.

Fifth, out of the veritable *dozens* of postnatal, prospective cohort studies of lead and ADHD that satisfy temporality and were relied on by Plaintiffs' experts (*see e.g.*, Def. Ex. 7, Gardener Rpt. at 64-78), Defendants invoke *a single one*—Ji, et al. (2018)—and distort the findings of the study beyond recognition. *See* Def. Br. 3 at 45-46. Defendants do not quote any portion of Ji (2018) because the study provides significant support for the opinions of Plaintiffs' experts. As the authors note: "Elevated early childhood blood lead levels increased the risk of ADHD…accumulating evidence has revealed that ***even low-level lead*** exposures still have adverse effects on neurodevelopment…more recent research lends even further support for the CDC guidelines that there is no threshold for the adverse health effects of lead exposure." Def. Ex. 91, Ji et al. (2018) at 2, 7 (emphasis added). And, Defendants' claim that "no association" was observed with blood levels of less than 5 ug/dL is false. The authors concluded that "the adjusted OR for children with 2-4 µg/dL was…1.08, 95% CI (0.81, 1.44)" *Id*. at 5. This is a positive association albeit not statistically

significant. As for "not controlling for genetics," as Plaintiffs' experts explained, it is proper to control for a confounder only when the variable is associated with both exposure and outcome, i.e. a gene that is associated with both an increased risk to metal exposure and ASD. *See* Ex. 89, Gardener Vol. 1 N.C. Dep. at 235:8-236:6. Defendants do not identify any such gene. Indeed, in the absence of such a gene, "genetics are not really a variable that would make the association between the heavy metals and the outcome itself spurious." *Id*. at 236:1-6. Finally, the other variables that were not adjusted for in Ji (2018) were controlled in other lead/ADHD studies, of which the overwhelming majority found an association. *See* Def. Ex. 7, Gardener Rpt. at 72-73.

Lastly, Defendants claim that Plaintiffs' experts' reliance on studies that show positive but not statistically significant results renders their opinions unreliable. *See* Def. Br. 3 at 46-47. This is contrary to both science and law. As Plaintiffs' experts explain, statistical significance is not determinative of reaching causal opinions. *See* Ex. 89, Gardener Vol. 1 N.C. Dep. at 158:4-159:15, 208:13-209:11; *see also* Def. Ex. 7, Gardener Rpt. at 9-10, 14-15 ("[I]t is inappropriate to disregard the relevance of any result merely because of its p-value."); *accord* Ex. 24, Ritz Vol. 1 N.C. Dep. at 113:19-114:9, 327:10-24. This is in keeping with good scientific practice, as recognized by the California state court judges who rejected this very argument by Defendants when assessing the admissibility of Plaintiffs' experts' methodologies, and the Ninth Circuit when assessing the issue in other litigation. *NC*, 2022 WL 21778549, at *12 n.11, 21–24 (rejecting Defendants' argument that Plaintiffs' experts failed to consider statistical significance or that statistical significance is a prerequisite to causation); *see also Hardeman,* 997 F.3d at 965 ("[E]ven where adjustment for other pesticides resulted in loss of statistical significance, the results still showed a positive association…Thus, contrary to Monsanto's criticisms, the general causation expert opinions were sufficiently supported by reliable epidemiological evidence[.]").

CONCLUSION

For the foregoing reasons, Defendants' Rule 702 motions should be denied.

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Respectfully submitted,

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CERTIFICATE OF SERVICE

I certify that on October 25, 2025, I electronically filed the foregoing with the Clerk of the Court using the ECF system, which sent notification of such filing to all counsel of record.

/s/ Aimee H. Wagstaff